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## COMMUNICATIONS

# THE CAUSE AND RESULTS OF OBSTRUCTION OF THE CENTRAL ARTERY OF THE RETINA: A STUDY OF ELEVEN CASES

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### Introduction

GEORGE COATS,[4, 5] (1905 and 1913), in reporting on three cases of obstruction of the central artery of the retina which he had examined pathologically, collected all those published in which a pathological examination had been made, and showed that, while the majority were best explained by disease of the vessel wall, with or without thrombosis, two of his own cases were undoubtedly embolic in origin.

There is no difficulty in understanding the onset and most of the phenomena of obstruction if gross disease of the artery wall is present, but a large proportion of the cases occur in young adults whose arteries must be presumed to be healthy. It is in these that pathology is of little assistance, owing to the absence of opportunity for obtaining the eye. Only two of Coats's collected cases were under 40 years of age and none was under 30 years. The value of the specimens, moreover, was inevitably diminished by considerable delay between the onset of obstruction and the pathological examination.

It is generally admitted that, in acute obstruction, as Coats[4] (1905) pointed out, three processes, in combination or separately, may be responsible :—

1. Embolism.
2. Endarteritis.
3. Thrombosis.

In recent years a fourth process, spasm of the arterial wall, has been put forward as the most satisfactory explanation of certain types of case.

Regarding the relative importance of any one of these processes, there appears at present to be no unanimity of opinion, excepting, perhaps, in cases occurring in later life in which arterial disease is known or may be presumed to be present. Here, endarteritis with thrombosis, is usually considered to be the cause.

The diagnosis between these possibilities may be greatly assisted by the history and by the general physical condition of the patient but, as Reimar pointed out, it must chiefly depend on the changes observed in the retinal vessels themselves, changes which vary from case to case and from day to day and are difficult both to interpret and to classify.

The literature consists largely of descriptions of cases and comments upon particular phenomena observed, but most are chosen for publication on account of some point of interest or obscurity and are, for analytical purposes, unsatisfactory. The terms used, moreover, are sometimes ambiguous. For example, words such as empty or collapsed, so frequently employed in reference to the ophthalmoscopic appearance of vessels, are too dogmatic. In encountering them, the suspicion that the vessels were neither empty nor collapsed is sometimes justified and an important observation thereby invalidated.

For the above reasons, a statistical examination of reported cases would have only limited value. The sex, however, and the age, excepting two cases recorded on account of their unusual youth, may be considered unaffected by selection, and I have analysed these two points in 51 cases in which the information is supplied.

The cases upon which my thesis is based are not selected, but consist of all those treated at Moorfields Hospital during the eight months February to September of 1928, making an incidence of 1 in 2,549 patients. Five of them were seen within 24 hours of the occurrence of obstruction. For permission to publish my observations on these cases, I wish to record my grateful thanks to Sir William Lister for cases Nos. 1, 6, 8 and 9, to Mr. Foster Moore for case No. 7, to Mr. Juler for cases Nos. 2 and 5, to Mr. Goulden for case No. 4, to Mr. Whiting for case No. 11, to Mr. Doyne for case No. 10, and to Mr. Scott for case No. 3.

I propose, firstly, to consider the history and general condition of the patients and then the changes seen in the eye, referring, as I proceed, to observations and opinions recorded in the literature. Lastly, I shall discuss the effects of arterial obstruction on the nutrition and function of the retina.

As I shall, where possible, discuss the cases collectively rather than individually, I have put the case notes together as an appendix and shall refer to them by number.

ANALYSIS OF AGE AND SEX.

| | *In 51 recorded cases* | | *In own cases* | |
|---|---|---|---|---|
| | *Male* | *Female* | *Male* | *Female* |
| Under 10 years ... | 1 | 1 | 0 | 0 |
| 10—20 years ... | 2 | 9 | 1 | 2 |
| 20—30 years ... | 3 | 5 | 0 | 3 |
| 30—40 years ... | 9 | 8 | 1 | 1 |
| 40—60 years ... | 3 | 3 | 1 | 0 |
| Over 60 years ... | 5 | 2 | 2 | 0 |
| | 23 | 28 | 5 | 6 |

There is substantial agreement between the two sets of figures and three points emerge.

(1) Taking the figures as a whole, the two sexes are about equally affected.

(2) In young adults it is more common in females and in later life more common in males.

(3) It is as common in young adults as in later life.

## Physical Condition

Four cases had mitral disease, two mitral and aortic, two aortic disease alone, and three revealed no evidence of cardiac abnormality. All the cases with cardiac disease were well compensated excepting No 5, and the patients professed to be in good health. Those with apparently healthy hearts were aged 16, 21 and 52 years. The urine in all cases revealed no albumen or sugar. In cases 2 and 8 a Wassermann reaction was done and found to be negative. The blood pressure was low in the imperfectly compensated case, No. 5, but in other cases was not abnormal. In the female cases the menstrual periods were normal.

The large proportion with endocarditis points to embolism as the cause. That many cases occur in young people without any sign of endocarditis is a well recognised fact, and it is possible that my series is unusual in the small proportion (2 in 7 under 40 years) shown. I shall return to this question when dealing with spasm.

### History

The onset was sudden in all cases except in the bilateral one, No. 11.

There was a history of a temporary obscuration in this bilateral case. No other case admitted having had previous obscurations or trouble of any kind with the eyes. The obstruction seems to have occurred at various times without reference to any particular activity. Three cases noticed the loss of vision on waking in the morning, but it is doubtful whether any significance can be attached to such a small proportion. They were all young patients with cardiac disease, and it is possible that strains thrown on the heart during disturbed sleep (MacWilliam) favour the production of emboli.

The history of case No. 1 is important. At first the whole field of vision was lost, but later the upper half returned. This is a very common finding in obstruction of one division of the central artery. Benson, Schnabel and Sachs, Hird and Harrison Butler report cases, and consider them to be embolic. There can be little doubt that, in case No. 1, the obstruction was caused by an embolus, firstly at or before the bifurcation and then, after an interval too short to affect vision permanently, further on in the branch where it was found. Such a history has a bearing on prodromal attacks and I shall refer to it again later.

## THE RETINAL VESSELS

The information derived from the vessels is very perplexing and has led to much uncertainty. To some extent this may be due to the fact that most patients are not seen until some days after the onset and then only at intervals of several days or weeks. I shall discuss the changes under the following headings and shall then shortly analyse the features presented by individual cases.

1. The circulation.
   - (*a*) The calibre of the arteries.
   - (*b*) Pulsation.
   - (*c*) Beading of the blood column.
2. Appearances of obstruction.

I shall refer mainly to the arteries as no changes of importance, except beading, were observed in the veins.

### The Circulation

It is a remarkable fact that some circulation is always retained or regained. If it stops entirely, as in Case No. 10, it soon returns to some extent, and it is rare to see stagnant beaded blood in all the main branches. This point was discussed at length by Coats[4] (1905), who concluded that it was best explained by anastomoses

between the retinal and ciliary arteries around the disc, a suggestion first made by Knapp. Schnabel and Sachs, on the other hand, argued against this and favoured incomplete embolism without thrombosis. Reimar discussed all the possibilities and found none satisfactory.

(*a*) *The calibre of the arteries.*

A small lumen indicates a restricted flow, but, as Parsons has pointed out, a normal lumen is no indication of the amount of circulation, for this depends also on the rate of flow which can only be seen when the red cells have aggregated. In the production of constrictions, the blood supply, the nutrition of the vessel walls, the intraocular pressure and the constrictor mechanism all may play a part and it is impossible to determine the importance of any one factor. Observation of a long series of cases might enable some classification to be made, but, until it is possible to examine pathologically several eyes of suitable cases soon after the onset, there will probably be no satisfactory solution.

The constrictions seen in advanced retinal arterio-sclerosis cannot be considered to bear on those seen in young people after acute obstruction. That there is not necessarily any constriction where a visible obstruction is present is shown in cases Nos. 1 and 2 (drawings).

In many of my cases, constrictions of small branch arteries occurred at some stage. These could always be explained on the grounds of defective blood supply. When red cells are clumped together they will naturally tend to keep to the main channel as long as possible. Cases Nos. 5 and 6 showed local constrictions on the disc. Case No. 5 was of a type sometimes described (Knapp, Benson, Lawson, Reimar) in which the arteries, small on the disc, widened out near the disc margin. This phenomenon has been referred to as evidence of cilio-retinal anastomoses (Coats[4] (1905), Parsons), but Schnabel and Sachs and Reimar thought it due to disease of the vessel walls. This patient was aged 19 years, so disease of the vessel walls was exceedingly unlikely. If it were due to anaemia of the walls alone, it would scarcely extend so frequently only to the disc margin. It is not a common feature of retinal arterio-sclerosis. It appears to be a late phenomenon—11 days in case of No. 5—and while it may be explained by assuming a small central circulation from incomplete obstruction or anastomoses within the nerve, and further anastomoses at the disc margin, the fact that the enlargement of the inferior division in this case occurred well inside the disc margin suggests that some other factor is responsible, possibly an active localised spasm secondary to embolism.

Case No. 6 is more difficult to explain although, up to a point, it resembles No. 5. As the superior division was much more affected than the inferior, the obstruction would appear to have been at the

main bifurcation just out of view within the nerve head. The corkscrew-like appearance of the blood column in the superior division, when first seen, was probably due, as in case No. 9 mentioned later, to slowing of the blood column and aggregation of the red cells. After a week, it would seem that the remaining circulation diminished on the disc, but was augmented by anastomoses at the disc margin (v. drawing). On the 8th day, fresh anastomoses in the nerve enabled the artery on the disc to fill again, but anaemia began to affect the superior nasal artery (v. drawing). A week later, the inferior division was found to be almost thread-like and conducted no blood. It was perhaps affected by secondary thrombotic changes at the bifurcation, but the absence of anastomoses is unexplained. As in case No. 5, it must be admitted that late secondary spasm of the vessel wall provides an alternative and less complicated explanation, though it is difficult to understand why a spasm should pass off in the superior division, in which the circulation was most defective, and obliterate the inferior division. This case (v. drawing) illustrates a point which I have frequently observed in the branch arteries in obstruction, namely, that when the blood supply to a branch is so severely limited as to cause the vessel to contract almost to obliteration, the contraction begins proximally at the junction with the main stem. It might be argued that an active localised spasm constricts the vessel and impoverishes the blood supply, but I do not think that the question of spasm need be introduced. I explain the phenomenon by supposing that, when the circulation in a branch slowly fails, the intraocular pressure blocks the capillaries supplied by it and prevents outflow. Blood no longer enters the vessel but tends rather to be withdrawn by the beaded blood passing along the main channel. The pressure in it therefore becomes greatest at the periphery and contraction occurs first near the parent artery.

(*b*) *Pulsation.*

When the circulation is not interrupted, the main arteries can be seen to pulsate vigorously when pressure is applied to the globe. Knapp first noticed the absence of pulsation when there was obstruction. Presence of pulsation implies that there is no gross obstruction to the blood flow, and its absence is evidence of definite obstruction, but is no guide to the extent. Rapid refilling suggests that a blood current is still present.

In case No. 9 it was very difficult to make the main arteries collapse on applying pressure to the globe. I interpret this as indicating sclerosis of the vessel wall.

(*c*) *Beading of the blood column.*

Beading of blood in the arteries and veins has been studied by Friedenwald. It is often seen in the veins, less commonly in the

arteries. It appears to be an aggregation of the red cells due to slowing of the circulation and is, to some extent, an index of the rate of circulation. A moderate slowing will cause beading in the veins only, a more marked slowing will cause it in the arteries as well. Also, when the circulation is very slow, there are clear cut beads, when it is faster, only small granules are with difficulty seen. Often, when there is a defective circulation as determined by absence of pulsation, there is no beading at all.

That the circulation may still be present without pulsation and without beading is shown by case No. 8. On first examination, the arteries were of almost normal calibre and appearance and showed neither beading nor pulsation on pressure, except in the healthy cilio-retinal artery which pulsated freely. A week later, without any other change, the first macular branch of the superior temporal artery was seen to contain fine granules of blood moving rapidly. The granules were difficult to see, but unmistakable. In the superior temporal artery no granules were seen, but a fair circulation, too rapid for aggregation, must have been present. That absence of beading does not necessarily mean that circulation is present, is shown by the normal appearance of the artery distal to the obstruction in case No. 1.

A curious appearance was seen in case No. 9 (v. drawing). The blood column was slowed and beaded as it emerged from the disc, but the arteries were of nearly normal calibre. In the superior division the blood flowed in a corkscrew-like manner without any segmentation until the next bifurcation. The outer wall of the artery was straight and unaffected, and the lumen was not responsible because the bends in the blood column were not constant. In the inferior temporal artery the blood column was even along the lower border of the lumen, but appeared very irregular above. That it was impossible to collapse the artery with reasonable pressure, showed that sclerosis was present and irregularities in the lumen possible, but the irregularities seen appeared constantly to change and may conceivably have been due to an effect of red cell aggregation.

## Appearances of Obstruction

Cases No. 1 and No. 2 showed clear-cut white areas in the arteries (drawings). There was no significant change in the diameter of the vessels, either as a whole or at the affected part. The only abnormality was an abrupt, clear-cut change from red to white, not a plasma-like whiteness but an opaque whiteness, with cessation of the circulation as determined by absence of pulsation on pressure.

In case No. 1 the obstruction slowly canalised (v. drawings) and pulsation returned, leaving only a spot of deep red in the vessel.

In case No. 2 it passed on, after massage, to a point in the inferior temporal artery where it remained a few hours before passing further out to the periphery (v. drawings). In this case also a mark of deeper red was left in the vessel. This appearance is seldom reported, and, in the literature, I have found only 4 definite cases amongst 51 (Reimar, Crisp, Harrison Butler, Mules).

Reimar's case (age not stated) had a thin stream of blood down the middle of the opacity. The stream widened and the obstruction disappeared. It had probably canalised when he first saw it. He considered that an embolus in a vessel should be obvious as such, and attributed the appearance in question to endarteritis of the posterior wall, so that, when the vessel filled again, the obstruction could not be seen.

Crisp saw his patient, a girl aged 14 years, on the day after the onset. The appearance, as drawn and described, was almost identical with that of case No. 1. Sometime between the 4th and 18th day the obstruction disappeared, leaving a narrow red strip across the vessel. There was no evidence of endocarditis. He called the condition spasm and, in spite of its normal size, regarded the vessel as being empty.

Harrison Butler's patient, a youth aged 21, was seen two and a half hours after the onset. The white area was at first bifurcation on the superior temporal artery and, peripheral to it, the blood was beaded and motionless. Five hours later the obstruction moved further to the periphery and gave the same appearance. Harrison Butler refers to the vessel being empty at the site, but the drawing shows the diameter unchanged. Endocarditis was present.

Mules's case, a girl of 21, was seen within an hour. The lower division to beyond the first bifurcation was of full size but white, with sharp demarcation between the white and the red portions of the artery. The eye was massaged at once and, on looking again, the artery was found to be normal. No cardiac disease was found.

These 6 cases may be diagramatically represented. The appearance undoubtedly represents an obstruction which either remains for a time and becomes canalised, or passes on to a point further along the vessel or out to the periphery. It cannot therefore be

an opacity of the vessel wall, nor can it be an ordinary thrombosis or patch of sclerosis. As the diameter is not affected, it cannot be a localised spasm. A vessel, moreover, especially when subjected to intraocular pressure, must accommodate itself to the volume of its contents, so, in these cases, can only be empty if flattened in one diameter only. But, if the lumen is empty, the elasticity of the artery wall should cause a general contraction. The appearance must represent the site of an embolus, but the whiteness is not easily explained. That the red cells can clump together suggests that it may be due to plasma with an increased content of leucocytes and platelets.

Rubert had the opportunity of examining pathologically a case of embolus (age and clinical details not given) 5 days after the onset, and found a homogeneous hyaline plug in the central artery in front of the lamina cribrosa. The artery wall was normal.

### The Cause of the Retained Circulation

There is anatomical, pathological and perimetric evidence for very small channels connecting the choroidal and retinal circulations in and around the nerve head. The appearance of such an anastomosis was seen in case No. 7 (v. drawing). There is also pathological evidence for embolism with incomplete obstruction and without thrombosis. (Coats[5] (1905), Schnabel and Sachs).

In my cases, if I exclude :—(1) those with probable endarteritis, and (2) those in which the obstruction appeared to have passed the bifurcation, cases Nos. 5, 6 and 8 remain for explanation.

In case No. 5, the calibre of the arteries was somewhat reduced and the circulation, although sufficient at first to lead to beading only in the veins and small arteries, gradually became more defective. The obstruction therefore did not pass on.

The artery supplying the healthy retina was probably not a ciliary vessel, but an early branch of the central artery. Clinical appearances suggested that it was slightly involved in the obstruction, which would therefore seem to have been in the region of the lamina cribrosa far enough back to give anastomoses full play. The gradual decrease in the circulation is best explained by supposing that the initial obstruction only gradually became complete during a week or 10 days, the final circulation being derived from anastomoses.

Case No. 6, referred to under " calibre of arteries," showed a fairly large volume of blood moving in the central artery at its emergence from the disc. Because :—

(1) The obstruction was probably at the bifurcation too far forward for effectual anastomoses to be present in the nerve.

(2) Only 24 hours had elapsed since the onset.
(3) The circulation gradually dwindled until, after two weeks, it had almost ceased.

It seems necessary to suppose that at first the obstruction did not occupy the whole lumen. The eventual restoration of circulation points to anastomoses.

Case No. 8 showed an absence of pulsation in main arteries of only slightly reduced calibre, indicating in this case, as I have shown, that the circulation, although obstructed, was too rapid for beading to occur. While it is probable that the obstruction was incomplete, four days had elapsed and it is just possible that anastomoses supplied the existing circulation.

### Summary Analysis of Individual Cases

Case No. 1.—The history and appearances, already discussed, point to embolism, firstly before the bifurcation and then just beyond it. The age and mitral stenosis confirm.

Case No. 2.—This was undoubtedly due to an embolus which stopped at the lamina cribrosa or main bifurcation and then passed on to where the inferior temporal vein crossed the artery, ultimately passing out to the periphery. The age and the mitral stenosis confirm this assumption.

Case No. 3.—The fact that the two small arteries which were unaffected and supplied the macula left the main stem shortly before the bifurcation, points to the obstruction having occurred just at the bifurcation. There was no such central obstruction when the case was first seen, so it must have passed on, probably down the inferior temporal artery which was most and longest obscured by oedema. Only an embolus could explain the findings. The age and the mitral disease are also in agreement.

Case No. 4.—The superior temporal artery only was affected. When the case was first seen, the obstruction had probably passed on to the periphery, as retinal pallor and a field defect were the only lesions found. This, with the age and presence of mitral stenosis, is best explained by temporary embolism.

Case No. 5.—I have referred to this case under " Calibre " and " Cause of retained circulation." The clinical observations, supported by the advanced cardiac lesions and the age, point to embolism.

Case No. 6.—I have referred to this case also under " Calibre " and " Cause of retained circulation " and concluded that the obstruction at first only partly occluded the superior division at the bifurcation, just out of view in the nerve-head. Later, secondary processes obstructed the inferior division. Embolism seems the most satisfactory explanation of this difficult case. It is supported by the age but not by the cardiac condition. Although the changes

in calibre suggest that secondary localised spasm may have occurred, nothing was observed at the first examination to warrant the view that the obstruction was originally caused by spasm rather than by embolism.

Case No. 7.—A healthy young girl with an apparently normal heart. Only the inferior division was definitely affected. Although the obstruction, as determined by beading and absence of pulsation, remained about a week, it could not be seen because the bifurcation into temporal and nasal branches occurred in the physiological cup and was obscured. As the whole of the inferior division was affected, this must have been the site of the obstruction.

The arteries were not greatly diminished in size yet the opportunity for effective anastomoses seemed small. The obstruction was probably, therefore, incomplete. The observations are best explained by embolism, but the cardiac condition is against this. A spasm would scarcely have remained confined to such narrow limits and have persisted for a week.

Case No. 8.—I have referred to this case under "Cause of retained circulation." It is uncertain whether the obstruction was partial or whether it was complete with good anastomoses. The obstruction remained and, when the patient left hospital two weeks after the onset, pulsation had not returned. The only evidence in favour of embolism was the well-marked mitral and aortic disease. There were some appearances in the other eye suggestive of old haemorrhages, and vascular disease cannot therefore be excluded.

Case No. 9.—The arteries on the disc were of fair calibre, but the circulation slow and beaded. The presence of arterio-sclerosis was suggested by the great difficulty in collapsing the arteries, and by the heart block. On the other hand, signs of aortic disease were present. Like No. 8 this is a doubtful case.

Case No. 10.—A complete obstruction lasting less than 24 hours in a man of 52 with an apparently normal heart suggests anaemia from a temporary fall of blood pressure in arteries narrowed by endarteritis. But, in this case, the arteries on the disc, ten minutes after the onset, were white and of about normal calibre, with complete stasis of circulation. The appearance, therefore, was similar to that in case No. 2, discussed under "Appearances of Obstruction," and, consequently, in spite of the lack of evidence of cardio-vascular disease, there is some reason for attributing the obstruction to embolism.

Case No. 11.—The obstruction was bilateral, complete and permanent. In a man of 65 with cardio-vascular disease, this is best explained by endarteritis with thrombosis. The more gradual onset and history of a prodromal obscuration differentiates this case from the remainder.

### Endarteritis

Endarteritis cannot safely be inferred in the retina from its presence elsewhere and is only obvious in its late stages. It does not follow that because vascular disease is present that it is the cause of the obstruction, and the changes in the vessels and circulation, as observed in cases 8, 9, and 10, do not enable a diagnosis of embolism to be excluded.

### PRODROMAL ATTACKS

Cases with prodromal attacks are probably less common than the number published would seem to imply. They are usually patients past middle life and are best explained by a temporary fall of blood pressure causing anaemia in arteries narrowed by endarteritis. (Parsons, Coats[4] (1905), Nettleship). Cases of embolic obstruction of one division only of the central artery which give a history of temporary complete blindness in the eye (case No. 1), or a case such as No. 2, undoubtedly embolic, in which a large part of the visual field was restored, show that an embolus can cause a temporary loss. Some prodromal obscurations may be caused in this way.

### SPASM

The question of spasm of the artery has been introduced to explain those cases which have prodromal obscurations and those in which no cardiac lesion is found. Bruner's case is one of the most convincing. There were as many as ten obscurations in a day and, during the attacks, all the arteries were reduced to mere threads. A thread-like appearance of the arteries occurring at once is an important point of difference between spasm and other causes. My cases showed that when an artery is obstructed by embolus no significant alteration in calibre occurs at an early stage. While, clinically, it may sometimes be impossible to make such a distinction, it is advisable to consider spasm as being of two types.

(1) Primary, occurring in cases in which there is no demonstrable arterial lesion.

(2) Secondary, occurring in consequence of arterial disease or injury such as embolism.

Primary spasm is a physiological possibility and can be induced in rabbits by injecting adrenaline into the vitreous (Redslob). It may be held to explain a certain small proportion of cases, but in the eleven under consideration nothing was observed to warrant its introduction.

It is noteworthy that the retinal artery is rarely, if ever, affected in Raynaud's disease. Secondary spasm offers a convenient explanation of the changes in calibre which occurred in cases Nos.

5 and 6. If Coats's theory that the retained field is due to anastomoses is a mistaken one and anastomoses at the disc margin do not occur, then these changes in calibre must be due to secondary spasm.

The retention of field is strong evidence for the presence of collateral vessels from the short ciliary arteries, such as the one seen in case No. 7 (drawing), but it does not necessarily follow that cilio-retinal anastomoses occur. The point must, for the present, be left open.

## EFFECTS OF OBSTRUCTION

### Changes in the Vessels

Cases Nos. 1 to 6 were last examined not less than two months after the onset of obstruction. These cases showed that when an artery has been obstructed long enough to lead to permanent changes in the retina, it ultimately becomes pale and narrowed to a size in appearance about one half that of normal, and not varying greatly from one case to another. Such an artery pulsates feebly on pressure. According to Parsons, this narrowing is due to the vessel accommodating itself to the volume of its contents by a thickening of its wall. In cases Nos. 5 and 6, the ultimate circulation was probably derived from anastomotic channels and may well have been of small amount, either on that account or on account of the loss of function of the retina supplied. The smallness of the circulation could therefore be explained as Parsons suggests. In cases Nos. 1, 2, 3 and 4, however, the obstruction passed on, leaving the arteries normal or nearly normal in size. It was only after some weeks or months that they became narrowed. In cases Nos. 3 and 4 only the superior temporal artery was affected and, in these two cases, loss of function of the retina supplied, as revealed by the visual field defect, could account for the ultimate smallness of the circulation. In cases Nos. 1 and 2, the field of vision showed only a partial defect and the explanation does not hold good. In these two cases, the smallness of the vessels must have been due to the nutrition of their walls having been permanently affected by the original obstruction, degenerative changes ensuing.

The fine white lines just outside and parallel to the vessels, occasionally reported (de Schweinitz), and noted in case No. 6 seven months after the obstruction, probably represent a peri-arteritis and peri-phlebitis which is best explained on nutritional grounds.

### Changes in the Retina

Pallor of the central retina is the chief objective characteristic of this disease. It is not certain whether it is due to oedema or to parenchymatous degeneration of the nerve fibre and ganglion cell layers. That the latter occurs, at all events in cases of permanent

obstruction, has been shown by Coats[4] (1905) who, however, considered that no case had been pathologically examined early enough to prove definitely whether the haze seen clinically was due to such degeneration or to oedema. In his second paper[5] (1913), he argued in favour of its being due to ischaemic necrosis. Rubert, on the other hand, in his case examined early, found oedema of the inner layers.

The appearance is well illustrated in Hamblin's drawings and has the following characteristics:

1. It is easily recognisable as early as two and a half hours after the obstruction (case No. 2). In the early stages it is most obvious in the neighbourhood of and overlying the main vessels, and appears in cirrus cloud-like striae which follow the general arrangement of the nerve fibres.

2. By about the second day, if the obstruction remains, a denser pallor which increases for several days is seen chiefly in the macular neighbourhood.

3. After a variable period of about 7 (case No. 3) to 14 (case No. 1) days, the haziness begins to disappear and the vessels show up more clearly. But the dense pallor in the central fundus remains for some weeks longer.

At this stage in case No. 6, in which there was no perception of light, spots of pearly, vesicular appearance were seen scattered over the central area. They were no doubt similar to the white spots noted by de Schweinitz, Reimar and Stephenson at a late stage and were probably a manifestation of necrosis of the inner layers. At this stage, also, the disc becomes pale.

4. The appearance of the macula varies within limits from case to case, but, except in rare instances, shows as a red spot which may be small with blurred edges or may be very clear cut and simulate a hole or haemorrhage. Case No. 6 was an example of the latter. That there was no hole was shown by the pearly vesicular spots extending on to the macula, and by the subsequent return to the normal appearance.

5. If a portion of the retina is supplied by a cilio-retinal artery, the healthy retina is in most cases sharply demarcated from the pallor, in contrast with gradual transition at the periphery.

6. Eventually the colour of the retina returns to normal.

Clinically the pallor is seen as two processes as described.

(1) An early haze associated firstly and chiefly with the vessels but extending over the central fundus.

(2) A subsequent denser central opacity which for a time consists partly of (1) but which remains as a clear white area after (1) has disappeared.

That the early pallor is not due to degeneration of the nervous elements of the retina is shown by:

(*a*) The early onset.

(*b*) The retained field of vision in cases Nos. 1 and 2.
In these cases some destruction of ganglion cells probably occurred locally, but not in areas corresponding to the entire distribution of the pallor.

Moreover, commotio retinae can cause a pallor equally dense, and a similar appearance along the vessels is sometimes seen, without any permanent change in the visual field, in retrobulbar neuritis and in cases of pressure on the optic nerve behind the globe. It is probably, therefore, due to oedema.

Following arterial obstruction, and against intraocular pressure, vascular dilatation cannot occur, but Lewis has shown that, in the skin, oedema is the result of an independent change in the wall in response to stimulation, increasing permeability, and is not a consequence of capillary dilatation. Whether the residual clear opacity is due to oedema or necrosis cannot be determined on the evidence available. The absence of haze suggests that it may be due to necrosis which, at this stage, may be presumed to be present.

## CHANGES IN THE VISUAL FIELD.

It is a common though not constant observation that, after complete obstruction, some vision remains which is limited to the perception of hand movements or, at most, the counting of fingers in the temporal field.

Hancock reported two cases in which the retained temporal field was definitely outside the 50° circle, and suggested that the nasal retina, which extends further forward than the temporal, is nourished at the periphery by the chorio-capillaris as well as by the retinal vessels.

Coats's[6] (1913) observations on 6 cases examined with tube fixation did not confirm this. He found that the retained field was around the disc, extending further temporally than towards fixation. He explained this as a consequence of the cilio-retinal anastomoses around the nerve head. de Schweinitz came to the same conclusion. As I have mentioned under " Spasm," such a field is evidence of collateral vessels, but not necessarily of anastomoses.

Case No. 5, in which the obstruction remained, confirms Coats's theory. Eleven days after the obstruction, some field contiguous with the blind spot was present for which the cilio-retinal artery could not have been responsible. Later, when fixation returned, the field was found to extend to about the 40° circle on the temporal side.

Similarly in case No. 8, in which also the obstruction remained,

some fixation returned on the 6th day and the field was found to be of the same type as that of case No. 5.

In cases Nos. 2 and 3, the obstruction passed on and some part at least of the restored field was due to the obstruction of some branches being, in time, so short as to enable the nutrition of the retina to be restored. It is noteworthy, however, that in case No. 3, four days after the obstruction and before the restoration of field took place, a narrow strip of percipient retina was certainly present on the nasal side of the disc.

In case No. 4, some restoration occurred by the disc but not elsewhere.

In case No. 10, half an hour after the onset of obstruction, some temporal field was present outside the 70° circle, but later all the peripheral field returned, leaving a central scotoma. The obstruction lasted only a short time and the case was therefore of the same type as No. 2. While it affords only doubtful confirmation of Hancock's theory, the latter is supported by the restored peripheral strip in case No. 1. The retention of a peripheral strip of percipient retina may be regarded as a more advanced state of the central scotoma type of field, discussed below. It is a theoretical possibility if the circulation returns before degeneration of the nerve fibres occurs, and if, in addition, two assumptions are made:—

(1) That nerve fibres are more resistant to anaemia than ganglion cells.

(2) That, at the periphery, the chorio-capillaris is capable of nourishing all the layers of the retina.

The greater resistance or better vascular supply of the nasal half of the retina may have reference to the greater phylogenetic age of these fibres.

Cases Nos. 2 and 3, in which the embolus passed on, require further consideration. In both, the field supports the ophthalmoscopic observation that the inferior temporal artery was the vessel most affected. In both, there is some general contraction of the field. In case No. 2, there is a central scotoma, and also in No. 3 there are indications that a central scotoma would have been present but for the unaffected macular artery.

The general contraction may be due merely to a raised threshold. That the restored retina in case No. 3 was not normal, was shown by the fact that, four months after the obstruction, the original healthy area could still be mapped out on the screen with a small object.

A central scotoma is rare and I have found only three cases recorded in which it was present. (Collins, Schnabel and Sachs, de Schweinitz). de Schweinitz suggested that it was due to pressure from oedema in the nerve.

In case No. 2, pressure in the nerve, sufficient to destroy fibres, would have caused considerable venous engorgement because the arterial obstruction was of short duration. No venous engorgement occurred, so there can be little doubt that local anaemia at the macula was the cause of the scotoma.

Consideration of the visual fields in these cases shows that degeneration need not occur, or that it may be local, causing a scotoma or sector defect, or may be general, causing complete or almost complete blindness.

The length of time that the obstruction persists is important, for, when it is short, the function of the whole or part of the retina may be restored. Alternatively, anastomoses may be too slow in forming or in accommodating themselves to the new requirements.

The position of the obstruction is also important, for, obviously, a peripheral injury may pass unnoticed, while obstruction at the bifurcation, as in case No. 6, cuts off anastomoses in the nerve and may lead to loss of all perception of light.

## THE MACULA LUTEA

Six cases illustrate the blood supply of the macula. The details may be tabulated as follows:

| Case No. | Unaffected artery. | Macula. | Vision First Examination. | Vision Last Examination. |
|---|---|---|---|---|
| 1 | Inferior temporal | Half supplied | 6/60 | 6/6 |
| 3 | Early branch of main artery | Supplied | 6/5 | 6/5 |
| 4 | Inferior temporal and? early branch of main artery | Half supplied | 6/18 | 6/6 partial fixation |
| 5 | Early branch of main artery | Not supplied | Counts fingers | 6/18 partly. Partial fixation |
| 7 | Superior temporal | Not supplied | Counts fingers | Counts fingers |
| 8 | Cilio-retinal | Not supplied | Hand movements | 6/18 |

The arterial distribution in case No. 7, in which the obstructed macular branch of the inferior temporal artery passed above the macula (Hamblin's drawing), was very unusual and made a return of vision unlikely.

It appears that:—

(1) The macula may be supplied by:
A twig from the main artery.
A cilio-retinal artery.
A branch from the superior or inferior temporal artery.
In part, by any of the above.
A cilio-retinal artery can be present without supplying the macula.

(2) Macular arteries are end arteries.
An anastomosis near the macula, made visible by beading of the blood, was seen in case No. 5, between the healthy branch from the central artery and the inferior temporal artery. It was probably a chance anomaly.

(3) If there is any healthy circulation in the immediate neighbourhood, a gradual restoration of some central vision may occur.

The central scotoma in case No. 2 shows that the macula is the most vulnerable area in retinal anaemia. In this case, the time during which circulation was absent can be fixed at between two and a half and five and a half hours. In Wood-White's case, the circulation was seen to return after an hour, and normal vision was restored. A similar case was reported by de Schweinitz, except that a small para-central scotoma remained.

The vulnerability of the macula may be due to:

(1) Precarious blood supply.

(2) Highly specialised nature.

(3) Late phylogenetic and ontogenetic development.

## CONCLUSIONS

### Cause

(1) While obstruction in later life may reasonably be interpreted on clinical and pathological grounds as a consequence of endarteritis, as explained by Coats, the most satisfactory explanation of the great majority of cases, not definitely so attributable, is embolism.

(2) There is evidence that embolism may occur in the absence of any other signs of cardio-vascular disease.

(3) There is evidence that emboli, when arrested, do not lead to early thrombosis, and may subsequently become detached and pass further along the vessel, with or without causing permanent damage to the retina. Also, that they may cause only partial obstruction.

## Results

*Arteries.*—While the ultimate reduction in size of affected arteries may, in some cases, be due to a process of accommodation resulting from diminished circulation, there is evidence that in others it is due to the obstruction affecting the nutrition of the walls, followed by degenerative changes.

*Retina.*—The retinal pallor is a combination of two processes:

(1) Oedema, lasting one to two weeks.

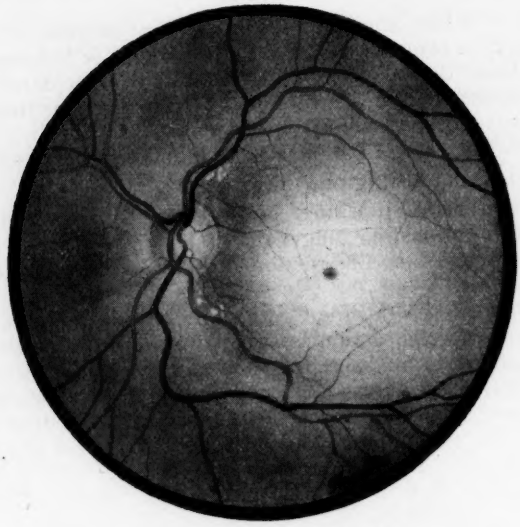
(2) Necrosis of the inner layers of the retina, commencing after and lasting longer than (1).

In cases of temporary obstruction of short duration, the former only may occur.

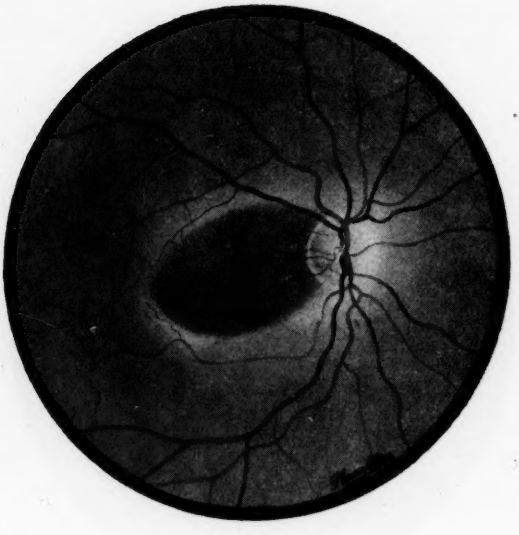
*The Visual Field.*—While Coats's observation that the retained field in complete obstruction is situated around the nerve head is confirmed, there is evidence that some field may occasionally be retained at the periphery. The character of any retained field depends on a combination of the duration, the degree and the position of the obstruction, and on the effectiveness of any cilio-retinal circulation or anastomoses. A central scotoma is evidence of a central obstruction of duration between, approximately, one and five and a half hours and is due to macular anaemia.

*Vision.*—The macula, owing chiefly to precarious vascular supply, is the most vulnerable region in retinal artery obstruction, but, if the duration of the obstruction is short or if there is some healthy circulation in the immediate neighbourhood, restoration of some central vision may occur.

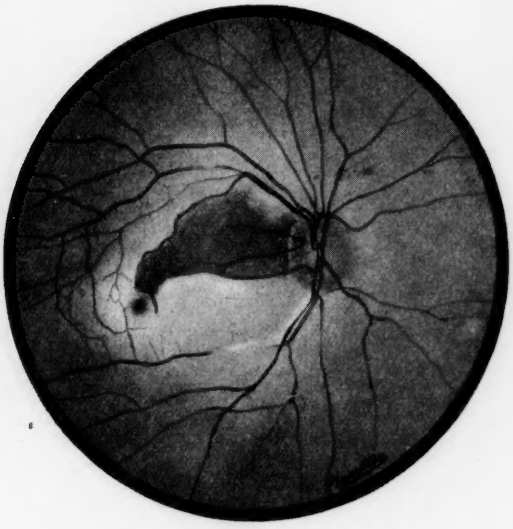
CASE NO. 1.



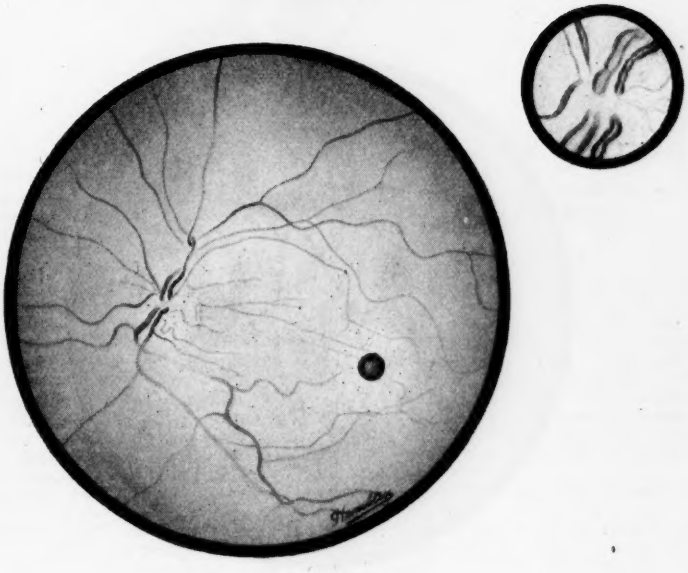
CASE NO. 2.



CASE NO. 3.



CASE NO. 5.



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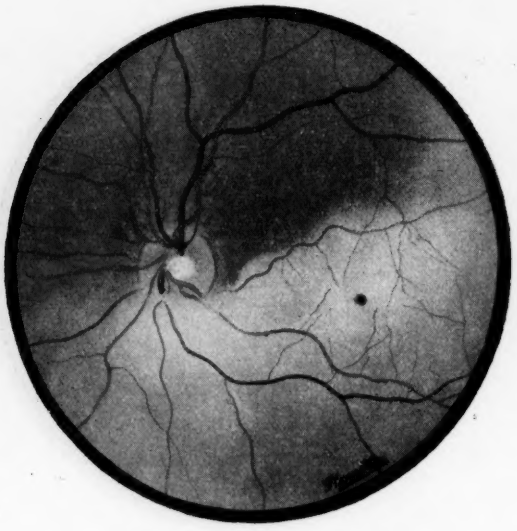

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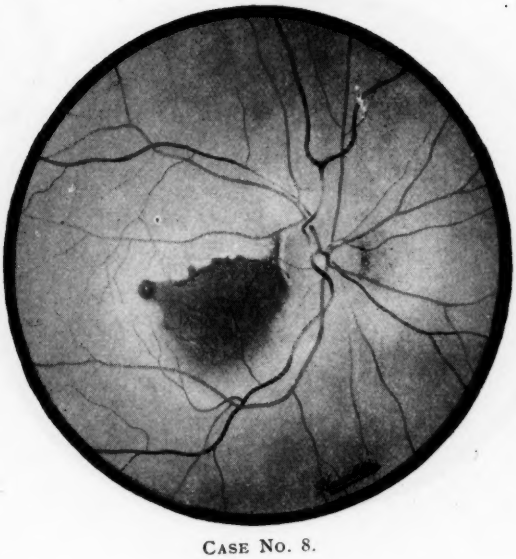


CASE NO. 8.

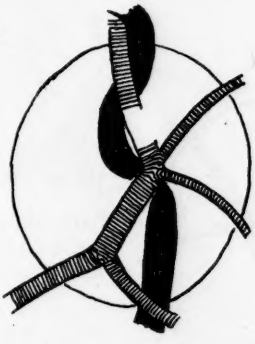
# APPENDIX

## Case Notes

CASE NO. 1.

G. B. Female. Aged 24 years. Married.

*History.*—Mid-day, 5 days ago, April 2, was washing her hands when, on looking down, noticed vision of R.E. misty. Is sure vision went suddenly. On day of onset whole field of vision of R.E. misty. Next day, she could see the upper half of objects only. Remained the same since. No previous obscurations or eye trouble. Always cold feet even in summer. Chilblains. Occasionally gets fainting attacks.




Gets " neuritis " in arms. Headaches across eyes since R.V. affected, not before. No history of tuberculosis. Periods normal.

*Condition.—General*: Healthy looking woman. Heart: mitral stenosis, not very advanced. B.P. 110/90. Urine: Sp. Gr. 1020. Acid; no sugar or albumen.

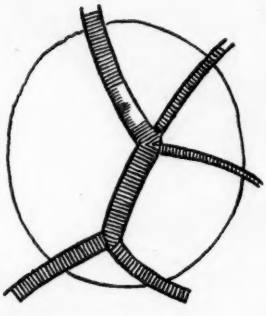
*Local*: Right eye. White. Cornea clear. No k.p. P. act. Media clear. Tension N.

April 7, 1928. Gross opacity of upper half of central retina shading off towards periphery but sharply demarcated below and not surrounding macula. Opaque retina slightly raised below, as determined by curve of small vessels. While the branches of the sup. temp. A. can be seen fairly clearly, a small vessel coming out from the disc is obscured. Although the central area supplied by the sup. temp. A. is most affected, there is marked oedema on the sup. nasal aspect of the disc, and to a less extent about the inferior vessels. A small portion (about $\frac{3}{4}$ disc diam.) of the sup. temp. A. on the disc is quite white; a clear cut section of the vessel. On pressure, all the main arteries except the sup. temp. pulsate vigorously. The sup. temp. A. does not pulsate at all. All arteries are of about normal calibre and no beading seen. Inferior div. of main vein larger than superior. Vision = 6/60, emmetropic. Sees only upper part of letter. Left Eye: White. Cornea clear. No k.p. P. active. Media clear. Fundus normal. L. Vision = 6/6.

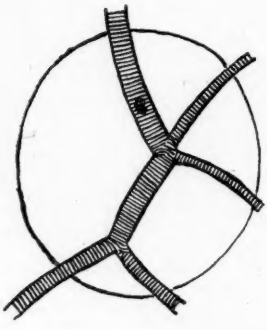
April 8, 1928. Segment of vessel still white and vessel does not pulsate on pressure. Other arteries pulsate on pressure.

April 10, 1928. White segment of artery now shows a thin streak of blood along its outer aspect as though the lumen were only partly blocked. Wall of vessel can be made out and there is no constriction or collapse. No pulsation on pressure in the affected artery.

10-4-28
Early canalisation



12-4-28
Pulsation on pressure returns.

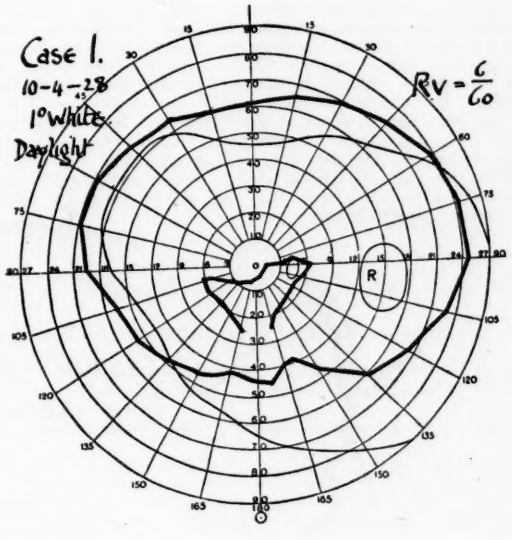


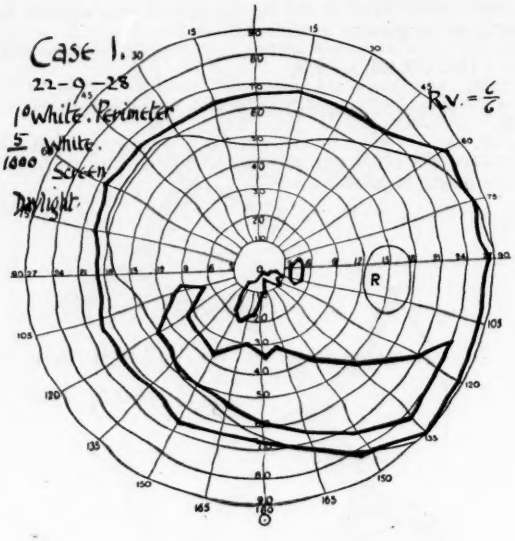
13-4-28
Red shot left

April 12, 1928. More blood is flowing through affected segment but lumen not filled. There is an irregularity in the restricted stream and at one point it almost widens sufficiently to fill the normal lumen. Sup. temp. A. now pulsates on pressure. R.V. with + 1 D.S. (dilated) = 6/18.

April 13, 1928. Artery now almost normal. Colour has returned completely, but at the site of the previous irregularity there is a spot of deeper red. Less oedema. R.V. = 6/18.

September 22, 1928. Fundus normal colour. Some temporal pallor of disc. Sup. temp. A. ½ normal size (same size as nasal branches). Inf. temp. A. normal. All arteries pulsate on pressure. R.V. = 6/6.





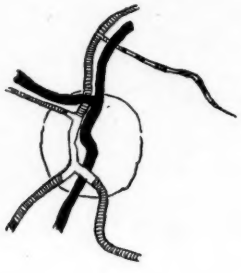

CASE NO. 2.

A. C. Female. Aged 38 years.

*History.*—Between 4 p.m. and 4.30 p.m. to-day, July 22, 1928, while riding in a bus, L.E. suddenly became blind. Never anything the matter before. No obscurations. General health good. Rheumatic fever when aged 13. No knowledge of heart trouble. Periods normal.

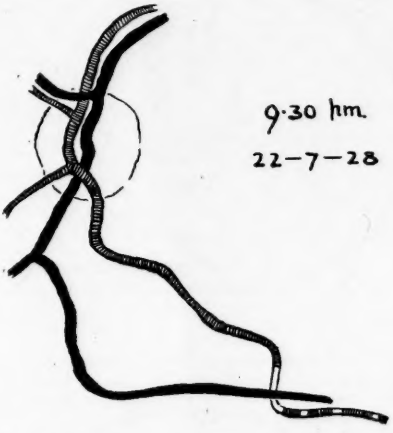
*Condition.—General*: Thin, pale woman. Heart: Mitral stenosis present. B.P. 115/75. *Urine*: No albumen or sugar. Wassermann reaction: Negative.

*Local*: July 22, 1928, 6.30 p.m. R.E. Healthy. R.V. = 6/6. L.E. White. Cornea clear. No k.p. Pupil inactive direct. Media clear. Central fundus pale. Cherry spot at macula. On disc, first parts of sup. nasal, inf. nasal and inf. temp. arteries white, the white part being sharply demarcated from the red. Beading in macular branch of sup. temp. A. L.V. = No perception of light. Treatment: vigorous massage. Amyl nitrite.



July 22, 1928, 9.30 p.m. Arteries on disc now red. Inferior temp. A. has a sharply demarcated white segment where it is crossed by inf. temp. vein. L.V. = Counts fingers.

July 22-23, 1928, 12 midnight. R.E. Normal. R.V. 6/6. L.E. Central fundus very pale, with cherry spot not very clearly defined at margins but standing out well. Striated oedema along main vessels. Veins normal. Arteries, normal size and main branches pulsate on pressure. Where the inf. temp. A. is crossed by the inf. temp.




vein the artery is definitely paler than elsewhere. The appearance is due to increased oedema, as it is quite different from that at 9.30 p.m. Across the artery here there is a narrow, darker red band. Far out at the periphery of the largest branch of the inf. temp. A., at a bifurcation, there is an obstruction beyond which the blood is beaded and motionless. Near the disc, alongside the sup. and inf. temp. vessels, there are some round woolly patches of pale exudate. L.V. = counts fingers in temporal field.

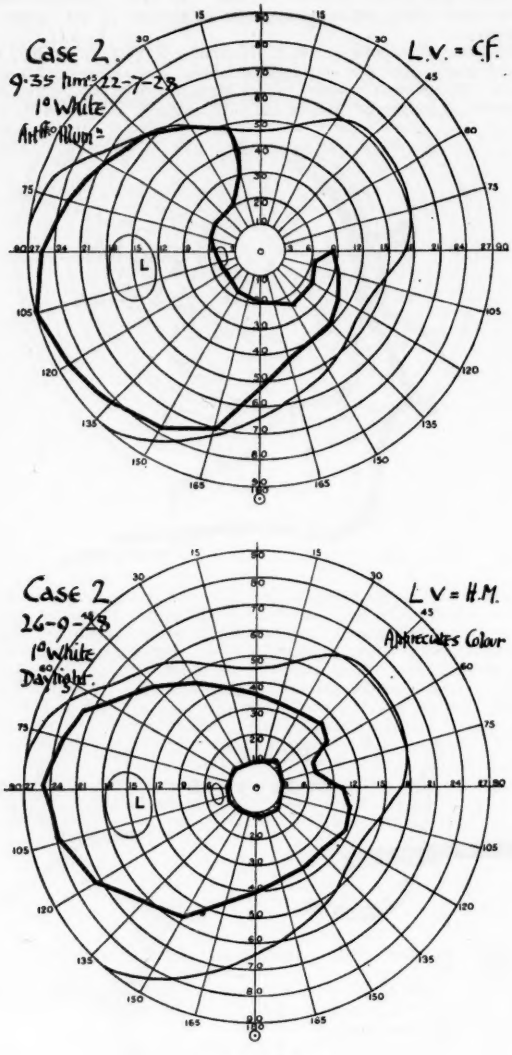
July 23, 1928. No obstruction or beading of blood seen anywhere.

July 24, 1928. No change.

July 28, 1928. Spot of red in inf. temp. A. has gone. Well marked radiating folds from macula. Vision unchanged. Central scotoma, with peripheral field complete but slightly contracted.

July 3, 1928. Oedema mostly along inf. temp. vessels. L.V. = counts fingers in temporal field.

September 26, 1928. L. Disc slightly paler than R. Sup. nasal and inf. temporal arteries a little irregular in size but no marked constrictions. Arteries all somewhat smaller than normal. Pulsate on pressure. Fundus normal colour. Macula normal. Tension N. L.V. = counts fingers. Positive central scotoma, with peripheral field complete but slightly contracted.




CASE NO. 3.

I. B. Female. Aged 21 years.

*History*.—Always good vision until 3 days ago when, on waking up, could see well in a very small field with R.E., but all around was black. Vision unchanged since. Was very tired and overworked night before sight went. No previous obscurations.

Sometimes feels faint and sick and has to lie down. Acute rheumatism 5 years ago. Does not mind the cold. No chilblains. Periods normal. Bowels regular.

*Condition.—General*: Healthy-looking girl. Heart: well marked mitral stenosis and aortic regurgitation. B.P. 110/60. Urine: No albumen or sugar.

*Local*: May 11, 1928. R.E. White. No k.p. Pupil sluggish, direct, normal consensually. Media clear. Pallor of central retina all around disc and macula, except the area between the disc and macula which is supplied by two small arteries coming from the main trunk before the bifurcation. This area is red. The macula appears to be supplied, but has the slightly granular appearance of the usual cherry spot. Retinal folds radiating from macula. Central artery and main branches pulsate on pressure. No obstruction seen. No beading. Vessels normal size. Periphery normal. R.V. = 6/5 slowly in a very small field. L.E. Healthy. Pupil active but poor consensually. Fundus N. L.V. = 6/5.

May 12, 1928. Much oedema covering vessels. Main arteries pulsate on pressure. No beading. Inf. temp. A. most hazy, but sup. temp. also very clouded for about one disc's diam. from disc. Field corresponds to unaffected area, but there appears to be some faint perception of movement in periphery of temp. field.

May 14, 1928. Beading noted in very small veins just above and outside macula. This is seen perhaps because there is a little oedema. Less central oedema.

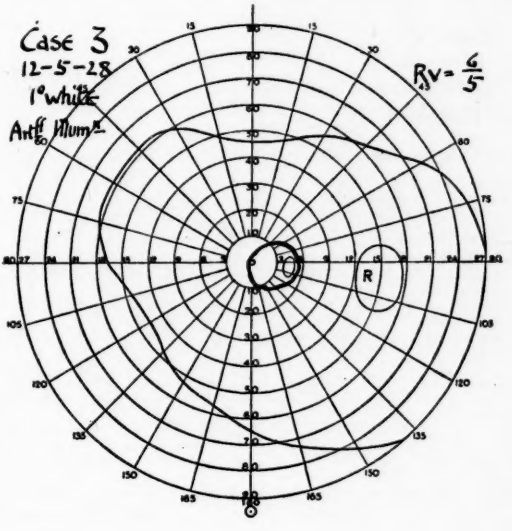
May 17, 1928. Central retina now a better colour and demarcation less noticeable. Disc pale. Inf. temp. A. smaller than before throughout its length. It does not seem to pulsate on pressure, perhaps on account of its size. No beading. Other main branches pulsate on pressure. R.V. = 6/5 slowly.

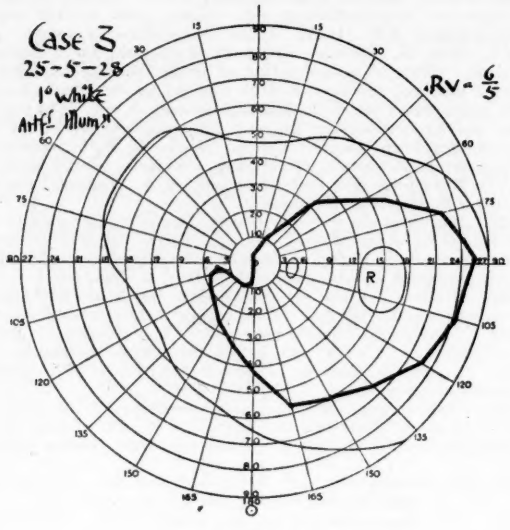
May 19, 1928. Oedema most marked around inf. temp. A. Inf. temp. A. pulsates on pressure. No beading. First temporal branch of inf. div. of cent. A. is definitely small in first disc's diam. of its length. Along sup. temp. vessels colour is almost normal.

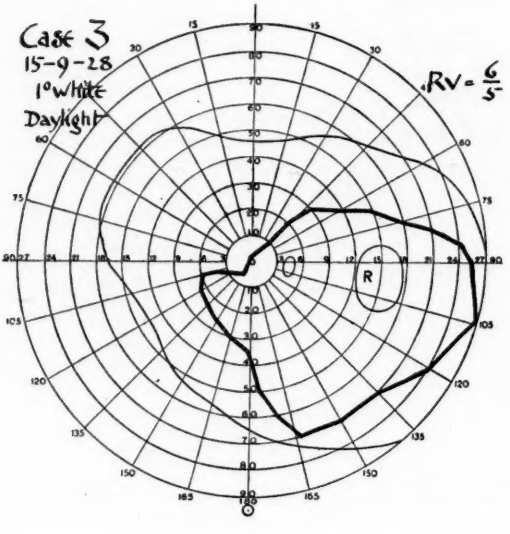
May 21, 1928. No oedema now except below and near disc. Retina normal colour above. Inf. temp. A. most blurred about a disc's diam. from disc.

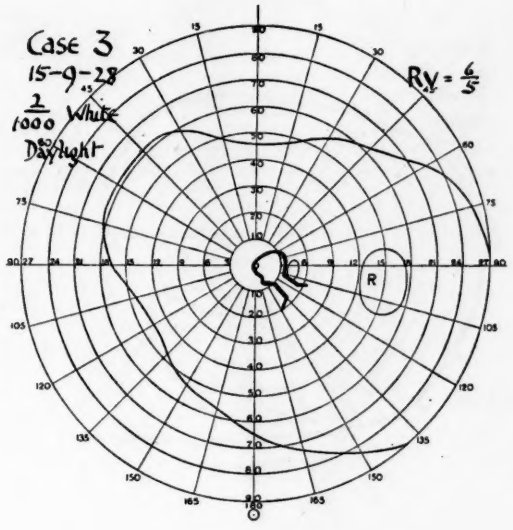
May 24, 1928. First temp. branch of inf. div. is smaller still. Scarcely any oedema. R.V. = 6/5 slowly.

September 15, 1928. Fundus normal colour. Disc pale. Main arteries small and pale. They pulsate on pressure. No beading. R.V. = 6/5.




Case 3
25-5-28
1° White
Artf. Illum.
RV = 6/5
R


Case 3
15-9-28
1° White
Daylight
RV = 6/5
R




CASE NO. 4.

F. V. Female. Aged 16 years.

*History.*—Five days ago awoke in morning to find vision of L.E. very misty. Now a little clearer. No previous obscurations or eye trouble. Has good health. Has been in hospital with chorea. Periods normal.

*Condition.—General*: Heathy-looking girl. Nasal obstruction. Heart: Mitral stenosis. Urine: No albumen or sugar.

*Local*: July 9, 1928. R.E. White. No k.p. Pupil reacts. Fundus and media N. R.V. with −3 D. Cyl. 180° = 6/6 partly. L.E. White. No k.p. Pupil reacts. Media clear. Area of white striated oedema extending from the disc along the sup. temp. vessels; extending down to, but not surrounding the macula. Sup. temp. A. is pale and slightly obscured by oedema but is not constricted. All main arteries pulsate on pressure. No beading. Macula prominent. L.V. with $\frac{+ 1.5 \text{ D.S.}}{- 4\text{D. Cyl. } 180°} = 6/18.$

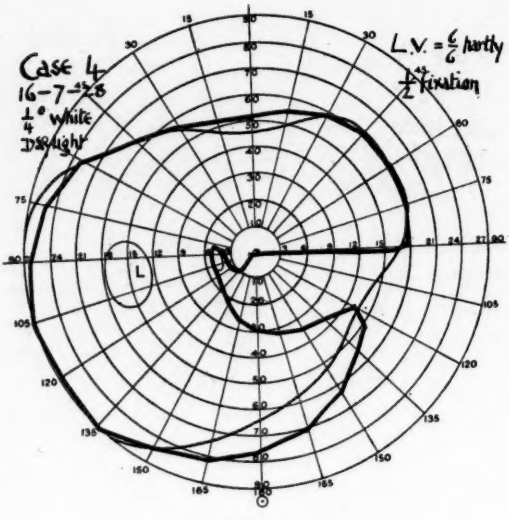
July 11, 1928. No change in appearance. No obstruction seen. L.V. with $\frac{+ 2 \text{ D.S.}}{- 4 \text{ D. Cyl. } 180°} = 6/6$ partly.

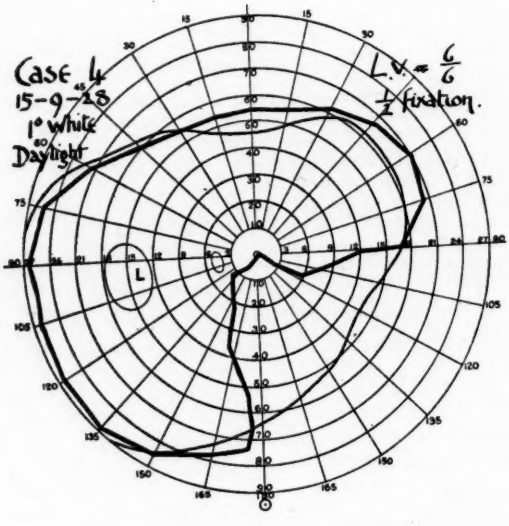
July 14, 1928. Radiating macular folds. No other change.

July 17, 1928. Fundus clearing. Only a little haze by disc.

July 18, 1928. Oedema almost all gone. L.V. with $\frac{+ 2 \text{ D S.}}{- 4 \text{ D. Cyl. } 180°} = 6/6.$

September 15, 1928. Superior temp. A. slightly smaller than inf. temp. A. Arteries pulsate on pressure. Disc normal. L.V. with correction = 6/6.


Case 4
16-7-45/28
¼° White
Daylight
L.V. = 6/6 partly
½ fixation


Case 4
15-9-45/28
1° White
Daylight
L.V. = 6/6
½ fixation.

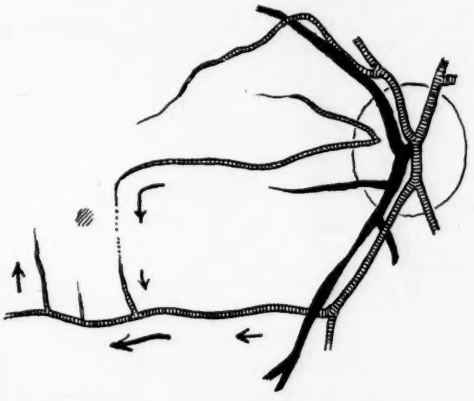
CASE NO. 5.

T. J. Male. Aged 19 years.

*History.*—This morning, 7 a.m., February 12, 1928, on getting up found R.E. blind. Never anything the matter before. No obscurations. Attended hospital for 3 years with heart trouble seven years ago. Sometimes feels faint, otherwise well.

*Condition.—General*: Pale and faint on attendance. Improved in 3 hours. Heart: Mitral stenosis marked. B.P. 85/45. Urine: Sp. Gr. 1028. Acid. No albumen or sugar.

*Local*: February 12, 1928, 10.30 a.m. R.E. White. No k.p. Pupil reacts slightly direct, well consensually. Media clear. Retina very slightly pale centrally, just sufficiently so to show up by contrast an area of healthy retina not quite extending to the macula, supplied by a branched artery emerging from the disc midway between the centre and the edge. Cirrus cloud-like striae of oedema along main vessels. Radiating folds from macula. Main arteries pale, but not constricted. Some small branches obliterated and appear as fine white lines. Arteries collapse on pressure.




Veins a little smaller than normal. Beading in some branches, granules in others. Slow movement. R.V. = counts fingers. L.E. Healthy. Fundus normal. L.V. = 6/6.

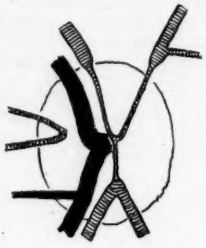
February 12, 1928, 2.30 p.m. R.E. Beaded blood still moving in veins. Both arteries and veins a little irregular in size and somewhat smaller than in L.E. Oedema increasing.

February 13, 1928. R.E. Veins vary greatly in calibre. Movement of blood still visible. Much more oedema. Vision unchanged.

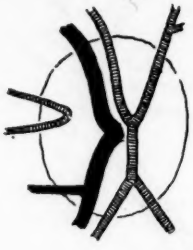
February 16, 1928. R.E. Movement of blood seen in some arteries as well as in veins. There is an obvious anastomosis between the lower branch of the cilio-retinal artery and the inferior temporal artery. Just as it passes the macula, movement of granular blood can be seen in the former moving peripherally. It can be traced down below the macula where, for a millimetre or so, it disappears. Then, in apparent continuity, an ascending branch of the inferior temporal artery appears and in it the same movement of blood can be seen moving centrally until the main artery is reached. In this and in its more peripheral branches the granular blood is moving peripherally (diagram). Dense central oedema, especially around vessels. Arteries a little smaller than normal and of more venous colour. Veins vary greatly in calibre.

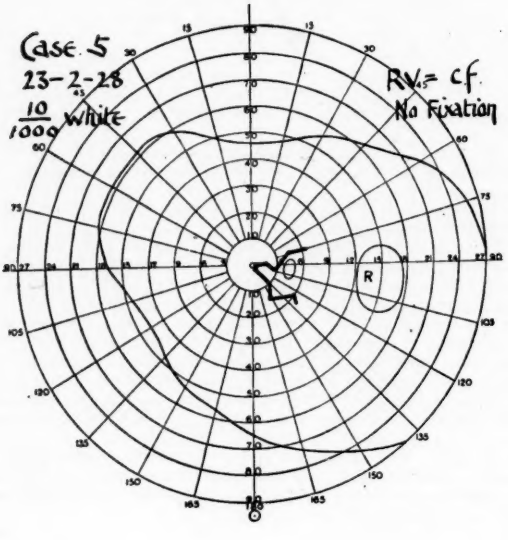
February 19, 1928. Less circulation and more branches obliterated. Main arteries small centrally. Arteries of cilio-retinal type appear affected. Beaded blood seen only where lower branch passes macula, but appearance is not quite normal. Small haemorrhage near macula. Vision unchanged.

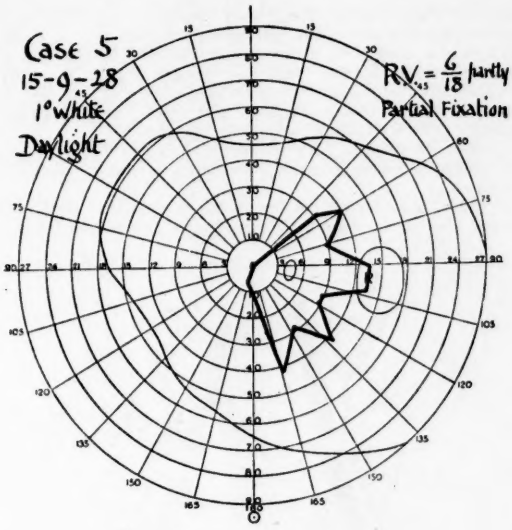
February 23, 1928. R.E. Until about disc margin, main arteries are like fine threads, they then open out to little less than normal size. Less oedema. Many fine haemorrhages by disc. Anastomosis still seen but less blood. Disc perceptibly paler than L.




September 15, 1928. Fundus normal colour. Disc pale. Arteries small and pale; they pulsate weakly on pressure. Veins small. R.V. = 6/18 partly.






CASE NO. 6.

G. A. Female. Aged 21 years.

*History*.—At work making beds yesterday morning, suddenly saw stars and found she could not see with the L.E. No previous eye trouble. No obscurations. Several attacks of tonsillitis and quinsy. In hospital for 6 weeks last year with pleurisy and pneumonia. Said to have "Kidney trouble." No chilblains or pallor of extremities. No fainting attacks. Headaches every few months and then sees two of everything. Periods normal.

*Condition.—General*: Healthy-looking girl of good colour. Heart: Not definitely abnormal but first sound suspicious. B.P. 130/90. Urine: Sp. Gr. 1015. Acid. No albumen or sugar.

*Local*: R.E. White. Cornea clear. No k.p. Pupil active. Fundus and retina normal. R.V. = 6/6.

February 6, 1928. L.E. White. Cornea clear. No k.p. P. active consensually. Media clear. Central fundus white and hazy. Arteries bright and red to just past

the disc margin and then hazy as though seen through a fog. Superior division has the appearance of uneven constrictions, giving resemblance to a corkscrew, as far as disc margin where it is crossed by vein. Artery narrower before than after crossing vein. Inferior division normal size until disc margin, then a little smaller. Veins small. Some branches of sup. temp. A. and a long branch of sup. temp. vein contain beaded blood. Cherry spot at macula large and very clear cut, simulating a hole. Periphery normal. L.V. = Doubtful perception of light.

February 8, 1928. No beading. No perception of light.

February 10, 1928. Some constriction at beginning of sup. division of A. Inf. div. larger than superior.

February 12, 1928. Superior div. as far as disc margin very small—almost thread-like. Thereafter, size is about normal. In the constricted portion a slow flow of

12-2-28.

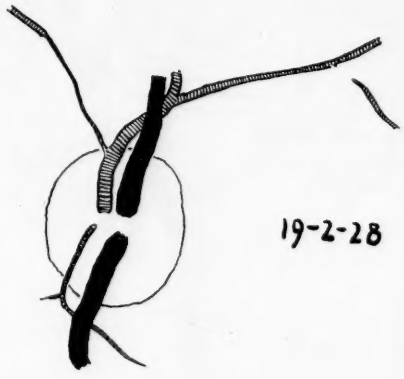
beaded blood without pulsations can be seen. No beading seen elsewhere. Inferior division of artery pulsates on pressure. Superior does not. Fundus very opaque. No retinal folds seen. L.V. = no perception of light.

February 13, 1928. Sup. div. of A. now large again. Same size as inf. div. Sup. nasal A. now constricted and thread-like at its origin. After about ½ disc diam. it fills out again.

13-2-28

February 19, 1928. Superior division not constricted. Sup. nasal A. thread-like for a disc's diameter. It is no larger than macular branches within a diameter of macula. No movement of blood seen in it. Inferior division very small—smaller

than macular branch of sup. temp. A. Inferior nasal and temporal arteries almost disappear, but the latter widens a little when it divides. The macular branch of the sup. temp. A. disappears for a short distance at its origin. Beads of blood seen in macular branch of inf. temp. A. While there is no progressive movement of the beads they move to and fro with cardiac systole. Beading seen in places in all parts of the fundus. No circulation seen anywhere. Veins nipped where arteries cross. Disc white. Haziness of fundus has disappeared, but dense central pallor remains. All over the macular region, both around and on the cherry spot, are what look like pearly dewdrops. Radiating folds from macula. Two small haemorrhages near inf. temp. A.




February 4, 1928. Optic atrophy. All vessels small. No movement of blood. Cherry spot larger. It and surrounding retina covered with white pearly spots of vesicular appearance.

September 22, 1928. All vessels small but contain blood. No local constrictions. Fine white lines run alongside arteries and veins outside walls. Fundus normal colour. Macula a little granular. L.V. = no perception of light.

CASE NO. 7.

K. C. Female. Aged 16 years.

*History.*—In the evening two days ago, noticed she could see only the lower half of objects with the left eye. Was swimming during the afternoon. No previous obscurations. Does not suffer from cold extremities; no chilblains. Very good health. Periods normal.

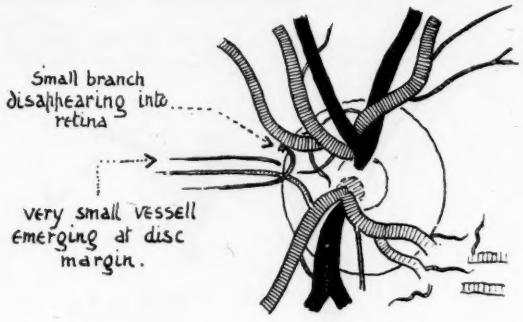
*Condition. General*: Healthy appearance. Heart: No evidence of abnormality. Urine: No albumen or sugar.

*Local*: R.E. Cornea clear. No k.p. Pupil active. Fundus and media normal. R.V. with glasses (—5.5 D.S.) 6/6.

September 29, 1928. L.E. Cornea clear. No k.p. Pupil active. Media clear. Intense pallor of lower half of retina, surrounding macula and obscuring vessels at disc margin. Pallor sharply demarcated above, fades gradually at periphery. Cherry spot at macula. Macular branch of inf. temp. A. passes above macula. Arteries of superior division normal and pulsate on pressure. Arteries of inferior division slightly smaller and do not pulsate on pressure. Some beaded blood seen in inferior arteries. L.V. = counts fingers temporally and above.

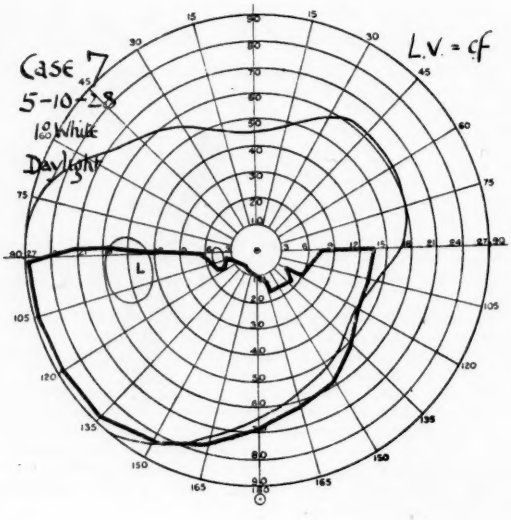
October 1, 1928. No beading seen.

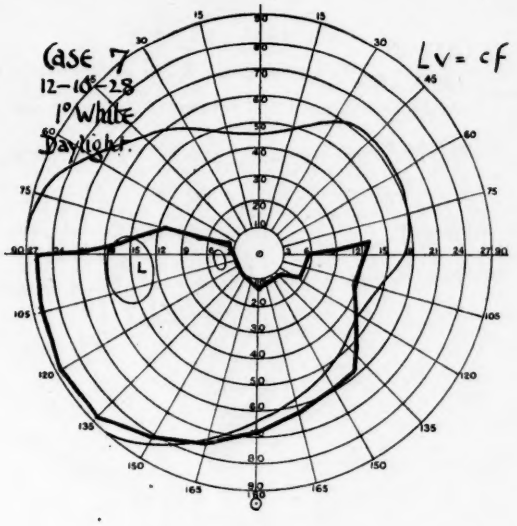
October 2, 1928. Owing to myopia and enlarged view of disc, several very small vessels can be seen on and around disc. From the superior nasal artery, a small branch is seen passing back through retina at the edge of the disc. Just below this, a fine arteriole emerges from the disc margin and runs out across the retina (drawing).




October 5, 1928. Oedema decreasing on disc. Small haemorrhage below disc. All inferior arteries now pulsate on pressure. Vision unchanged.

October 12, 1928. No change.






CASE No. 8.

S. F. Male. Aged 36.

*History.*—Four days ago, on return from work, he began to read and found R.V. misty. Is sure it was all right at work. Has remained the same since. Good vision in the army. No history of previous eye trouble or obscurations except occasional momentary mistiness when reading. Severe burns over chest and abdomen 1912. Discharged from army on account of heart trouble. No numbness or chilblains. No subjective cardiac symptoms.

*Condition.—General*: September 11, 1928. Healthy appearance. Heart: Well-marked aortic incompetence and mitral stenosis. Urine: Sp. Gr. 1010. Acid. No sugar or albumen. Wassermann reaction: Negative.

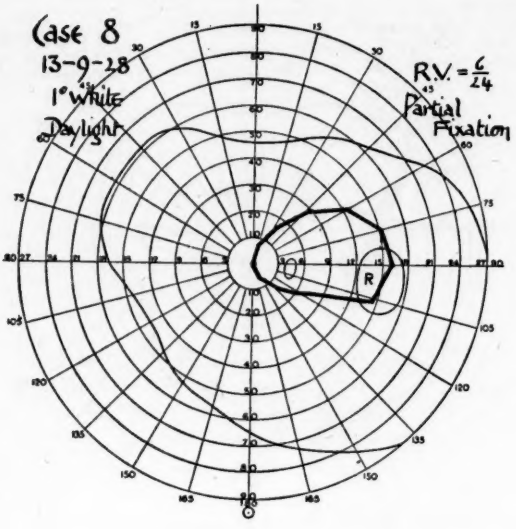
*Local*: R.E. Cornea clear. No k.p. Pupil sluggish. Media clear. Dense central retinal opacity with not much haziness over vessels. Area between the disc and macula and just up to macula healthy and supplied by a cilio-retinal artery. Macula slightly granular. Vessels all just slightly smaller than in L.E. but otherwise appear normal. On pressure the cilio-retinal artery pulsates, the main arteries do not. This is quite definite. No beading seen anywhere. No haemorrhages seen. Some light-brown pigment is present alongside the small vessels running from the sup. and inf. temp. arteries through opaque retina towards the macula. R.V. = perception of hand movements, temporal side. L.E. Cornea clear. No k.p. Pupil active. Media clear. T.N. Fundus shows normal vessels with faint irregular pale spots between disc and macula and along vessels. Perhaps old haemorrhages. Arteries pulsate normally on pressure. L.V. = 6/6.

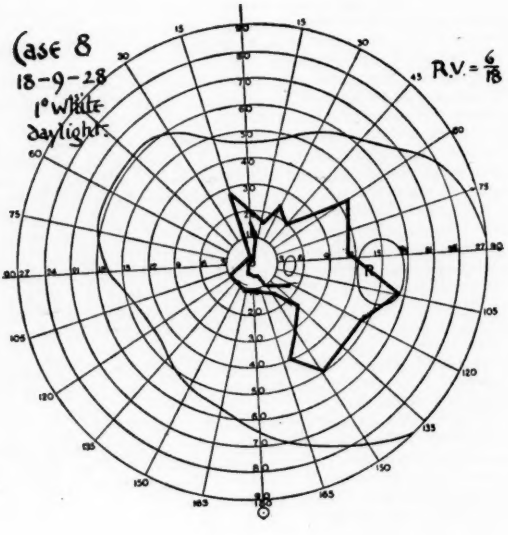
September 12, 1928. Vision improved. No fundus change. Absence of pulsation in arteries confirmed. Cilio-retinal A. pulsates. R.V. = one half of 6/60 letter direct. 6/24 when fixing just to the side.

September 15, 1928. No change in fundus. R.V. = 6/18.

September 18, 1928. First descending branch of sup. temp. A. now shows fine granules of blood moving peripherally without pulsations. First branch of sup. nasal A. thin just after origin for a disc's diameter. Fundus more opaque. No pulsation on pressure in main arteries. R.V. = 6/18.

September 21, 1928. No change.


Case 8
13-9-28
1° White
Daylight
R.V. = 6/24
Partial
Fixation
R


Case 8
18-9-28
1° White
Daylight
R.V. = 6/18

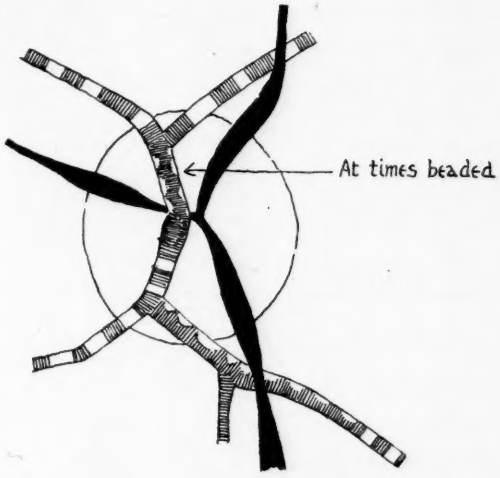
CASE NO. 9.

J. P. Male. Aged 70 years.

*History.*—Perfect sight R. and L. until this morning. Sight good on getting up. While dressing, slipped on hearthrug, but did not fall. On recovering balance, vision in L.E. completely gone. No previous obscurations. No children. Good health except for " asthma and bronchitis."

*Condition.—General*: June 5, 1928. Healthy appearance. Heart: Enlarged. Aortic systolic murmur. Sounds not very clear. Pulse 36. Complete heart block. Lungs: Emphysema. C.N.S. Normal. Urine: No albumen or sugar.

*Local*: R.E. White. No k.p. Pupil active. Media and fundus normal. R.V. with +1.5 D.S. = 6/6. L.E. White. Cornea clear. No k.p. P. sluggish. T.N. Media




clear. Very marked central retinal opacity. Macula is grey and surrounded by a lighter grey ring. Disc good colour. Superior division of main artery and sup. temp. A. smaller than normal but not collapsed. Beaded blood passes along these arteries in a slow, steady stream. As far as the disc margin the blood is sometimes beaded and sometimes has a corkscrew appearance. Inferior division and its branches larger than superior. Beaded blood passes only with each systole. In inf. temp. A. blood column is very uneven above, even below. The irregularity varies (drawing). A small branch comes off the main artery before the bifurcation, but it is affected. On pressure, arteries do not pulsate, nor do they collapse, except with considerable pressure. Stasis in veins. Calibre very uneven. L.V. = perception of hand movements in temporal field.

June 7, 1928. Movement of blood in superior division very difficult to see. Very obvious in inferior division as before. No pulsation of arteries on pressure. A great deal of pressure is required to stop the circulation. Some movement of blood seen in veins. A few very small haemorrhages near first part of inf. temp. A.

June 8, 1928. Vessels smaller. Blood almost stagnant but a little movement seen in large vessels. In small branches beaded blood not moving. No pulsation on pressure. L.V. = perception of hand movements in temporal field.

CASE NO. 10.

G. B. Male. Aged 52 years.

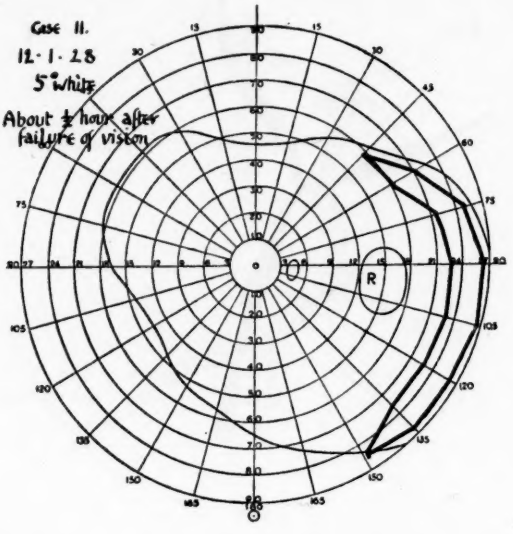
*History.*—Sudden failure of R.V. 10 minutes ago while walking along. No previous obscurations. Good general health.

*Condition.—General*: January 12, 1928. No sign of cardio-vascular disease.

*Local*: R.E. All main arteries white to about a disc's diameter from the disc and thereafter contain motionless beaded blood. Arteries on and near disc, although white, are of about normal size. Beaded blood in veins. Macula slightly accentuated. R.V. = perception of light. L.E. healthy.

January 13, 1928. Cherry red spot and faint surrounding oedema. Arteries of good calibre. No beading. R.V. = perception of light.

March 16, 1928. Vessels full. Full peripheral field with central scotoma.




CASE NO. 11.

A. M. Male. Aged 65 years.

*History.*—Had good sight until 9 days ago. Then, while sitting down, L.V. became dim but not blind. Better next day. Two days later both slowly became dim and he could not see even the light. No previous eye trouble. Had occipital headaches for 10 weeks. Otherwise satisfactory health.

*Condition.—General*: March 19, 1928. Heart enlarged. Aortic systolic murmur. Feeble arterio-sclerotic pulse. Lungs: Emphysema. Urine: Sp. Gr. 1020. Acid. No sugar or albumen.

*Local*: R.E. Cornea clear. No k.p. Iris atrophic. Pupil under mydriatic. Very dense opacity of central fundus. Cherry spot at macula. Arteries and veins small. No movement of blood seen. Arteries do not pulsate on pressure. Disc pale. Probably complete stasis and optic atrophy. R.V. = no perception of light. L.E.

Cornea clear. No k.p. Pupil under mydriatic. Iris atrophic. Less opacity of central fundus than in R.E., but dense. Cherry spot. Arteries of uniform size. All smaller than normal, but not so small as in R.E. No movement of blood seen in arteries. No pulsation on pressure. A little beaded blood seen slowly moving in veins. L.V. = no perception of light.

March, 23, 1928. No change.

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# AN UNUSUAL CASE OF CORNEAL DISEASE

BY

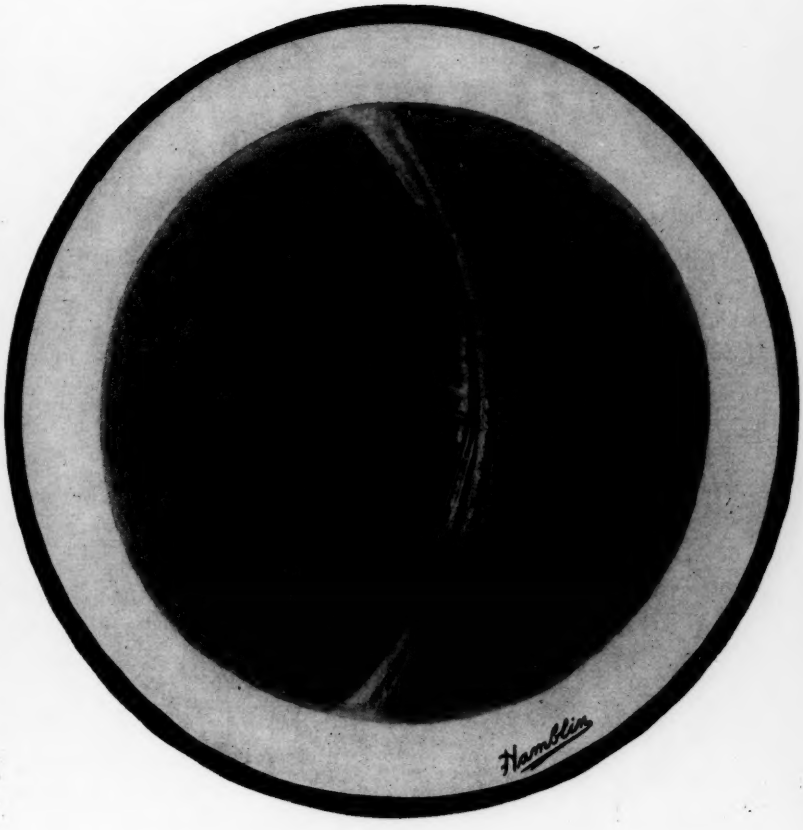
F. A. WILLIAMSON-NOBLE

LONDON

THE pathology of corneal dystrophies is not yet fully understood, and as the following case presents some points of interest, it was thought worth while to report it, particularly as the notes go back to the time before the disease developed.

The patient, a female domestic aged 44 years, was first seen as a refraction case at the Central London Ophthalmic Hospital in October, 1924. Under homatropine and cocaine the right eye with +1.0 D.sph.+1.0 D.cyl. 180° read 6/9 and the left with +1.5 D.sph.+1.0 cyl. 180° read 6/6 partly. She attended again in April, 1927, complaining that she saw a mist now and again with the left eye. Nothing abnormal was found in the eye, her refraction was practically unaltered and her vision the same as before. In November of the same year she complained of seeing a spider's web round lights with the left eye; she was seen on this occasion by the house surgeon and no further note was made except that vision had gone down to 6/18 with correction. I had an opportunity of examining her at her next visit, early in December when the slit-lamp showed a slight opacity occupying the pupillary area in the posterior third of the cornea. There were no traces of inflammation, no bedewing, no vascularisation or ciliary flush, no keratic precipitates or particles in the aqueous and no iris adhesions, also there was no sign of corneal anaesthesia. The Wassermann reaction was negative and no focus of infection could be found in the teeth, tonsils, nasopharynx or accessory sinuses. The vision was now less than 6/60 and the condition appeared to be some obscure dystrophic process which might quite reasonably be expected to attack the other eye. It was difficult to know what treatment to adopt in order to avoid this, and I had to proceed on what were rather empirical lines.

Three possible explanations occurred to me for the condition: (1) That it might represent the result of endocrine disturbance. (2) That it might be a deficiency disease—on the lines of keratomalacia. (3) That it was due to some defect in calcium metabolism. The patient was therefore put on to thyroid and parathyroid and given cod-liver oil and artificial sunlight treatment. Under this régime the right cornea remained clear and in March, 1928, she thought that the sight of the left eye was a little better though it was still less than 6/60. By the end of April, the disc of opacity in the posterior layers of the cornea was about the size of the undilated pupil, but mydriasis did not seem to improve vision.


Hamblin

Treatment was continued as before, potassium iodide being given intermittently and by June, 1928, the left eye could see 6/60 with the old glasses, though it relapsed to less than 6/60 in July. In January of this year, a star-shaped pigmented area had developed in the anterior layers of the cornea and a wrinkle had appeared in Descemet's membrane, the opacity being unchanged. The condition as seen with the slit-lamp is shown in the figure. In June it was thought that an optical iridectomy might be of service, but before doing this, I decided to go thoroughly over the patient's refraction again. To my surprise I found that the left eye with +9.0 D.sph.+1.0 cyl. 60° read 6/12, and one letter of 6/9—the opacity was by now rather faint, but still clearly visible with the slit-lamp and the change of refraction was evidently due to cicatricial flattening of the cornea following on partial resolution of the opacity.

A rough calculation shows that a diminution of 8 D. in the refractive power of the eye could be produced by a flattening of the cornea sufficient to increase the radius of curvature of its anterior surface from the normal 8 mm. to 9.74 mm.

The steps of this calculation are as follows:—the formula connecting refractive index, radius of curvature and focal length is $F = \frac{\mu R}{\mu - 1}$. In a problem of this kind, where we are concerned only with a small difference, we shall make no serious error if we regard the refraction system of the eye as being homogeneous. We must therefore first determine the value of $\mu$ for an eye, being given that F = 22.7 mm. (the distance from the anterior surface of the cornea to the macula) and R = 8 mm. - the radius of curvature of the anterior surface of the normal cornea. We thus have $22.7 = \frac{8\mu}{\mu - 1}$ which gives $\mu = 1.54$. A focal length of 22.7 mm. measured from the anterior surface of the cornea is equivalent to 44 dioptres.

The effect of the disease in the cornea was to reduce this by 8 D. and to make it 36 D., *i.e.*, 27.78 mm. Again using our formula $F = \frac{\mu R}{\mu - 1}$ and substituting this value and the value we have already determined for $\mu$ we get R = 9.74 mm.

The patient was examined with a Javal-Schiötz ophthalmometer which gave a difference of 4 dioptres between the two eyes, corresponding to an increase of 1 mm. in the radius of curvature of the cornea. This can be explained by the fact that the image of the mires is formed by the mid-peripheral and not by the pupillary area of the cornea. The opacity was limited to the latter area and the flattening would therefore be most marked in this area also.

Conclusions:—The interest of the case lies in the following points:—

1. The development of an opacity in the posterior layers of the left cornea unattended by any inflammatory reaction.

2. The restriction of this opacity to the cornea of one eye only; whether this was due to the treatment adopted, it is not, of course, possible to say.

3. The subsequent development of eight dioptres of hypermetropia, with an alteration of 60° in the axis of the astigmatism.

## B.WELCHII INFECTION OF GLOBE FOLLOWING PERFORATING INJURY

BY

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SENIOR HOUSE SURGEON, ROYAL LONDON OPHTHALMIC HOSPITAL

THE following case would appear to be sufficiently rare to justify its publication.

V. E., a labourer, aged 46 years, attended the Royal London Ophthalmic Hospital on May 4, 1929, at 1.30 p.m. He stated that on the previous afternoon he was chipping some concrete with a hammer and chisel when something flew up and struck his left eye. It caused him little trouble at the time, and he continued work. He attended because it still felt as if there was something in his left eye.

On examination the right eye was normal. The left eye was slightly injected, and there was a small perforation of the sclera 3 mm. outside the limbus. The cornea was bright and clear. Several small bubbles of gas were present in the anterior chamber, and during examination a fresh one was seen to come forward through the pupil. The eye was not tender. L.V., shadows.

5.30 *p.m.* The condition of the left eye was much changed. There was considerable pain in and around the eye. There was much chemosis and the cornea was no longer bright but oedematous and hazy. The anterior chamber was abnormally deep, the globe was very tender, and there was no perception of light in the eye. The patient felt ill, and had a "toxic" appearance. Temperature 98.2° F., pulse 90.

8.0 *p.m.* There was now marked oedema of the left upper lid, greatly increased chemosis, and some proptosis. A large bubble of gas could be seen, distending the anterior chamber and practically filling it.

8.30 *p.m.* Evisceration was carried out under general anaesthesia. A corneal incision was made, the bulbar conjunctiva being left intact. There was a definite crackling sensation on cutting into the anterior chamber and several small bubbles of gas appeared when the vitreous was being scooped out, but there was no pus. 25 c.c. of anti-gas serum (B. Welchii) were given intramuscularly and 10 c.c. were injected into the orbit through the lower lid.

The patient's temperature rose to 100°F. the day following the operation, but apart from that the temperature remained normal. He was discharged on May 15, having made an uneventful recovery. A stab culture was made from the contents of the vitreous at operation. From this a gas forming organism was grown identical with B.Welchii, of which it appeared to be a pure culture.

There are few cases described of panophthalmitis due to gas-forming organisms. James[1] describes two cases both similar to this in that they both had very acute intraocular infection, without pus formation, following quickly on the perforating wound. In both cases were bubbles of gas seen in the anterior chamber. One case was treated by excision and the other by evisceration. Both made a normal recovery.

Other cases are described by Oreste[2] and Chaillous[3]. In their series there is a note in each case that the patient was more ill than would be expected in an ordinary panophthalmitis, but there is no mention of gas formation. The B.perfringens was found in each case by routine bacteriological examination. In two cases hypopyon developed.

Lutz[4] also describes three cases in which a gas-forming organism was found on routine bacteriological examination, but all of them appear to have suffered from what was clinically a panophthalmitis of the usual type.

Ridley[5] recorded a case very similar to this one at the last Annual Congress of the Ophthalmological Society of the United Kingdom, the details of which are not yet in print.

I wish to thank Mr. F. A. Juler for permission to publish this case. The bacteriological examination was carried out by Dr. S. H. Browning.

## REFERENCES

1. **James, R. R.**—*Ophthal. Rev*, Vol. XXIX, p. 181.
*Trans. Ophthal. Soc. U.K.*, Vol. XXX, p. 179.
2. **Oreste.**—*Ann. d'Ocul.*, p. 35, 1910.
3. **Chaillous.**—*Ann. d'Ocul.*, p. 115, 1905.
4. **Lutz.**—*Klin. Monatsbl. f. Augenheilk.*, Vol. XLVIII, p. 31.
5. **Ridley.**—*Trans. Ophthal. Soc. U.K.*, Vol. XLIX.

## ANNOTATION

### Blindness in Animals in 1641

The 33rd volume of the publications of the Surtees Society, dated 1857, is entitled " Rural Economy in Yorkshire in 1641; being the farming and account books of Henry Best, of Elmswell, in the East Riding of the County of York." On page 79 occurs the following note on blindness in animals which may prove of interest to our readers.

" It is usuall with sheepe, and especially with hogges and lambes, to fall blinde by reason of a humour that falleth out of the head into the eye, wharby groweth (as it weare) a scumme over the stive of the eye; many shepheards will undertake to cure this by bleeding them in the wykes of the eye with a penne-knife, but the onely way is to take groud-ivy-leaves, and to chewe them in the mouth, and take out the leafe with the finger after yow have sucked the juice from it, this juice yow are to spurte into the eye morninge and eveninge, or if yow will thrice a day; and duringe the time of the blindnesse, if it bee in summer time, yow are to putte into some little place wheare it can neyther hang itselfe in briars nor runne into any water; if it bee in winter time, yow are (to putte) to some hey-mowe, and in a fortnight or three weeks it will eate of the scumme and the sheepe will see again."

None of the dictionaries at our disposal shed any light on the meaning of the words stive and wykes, but a possible cause of the blindness may be suggested in an attack of traumatic interstitial keratitis; we have seen an excellent example of this years ago, in a retriever, at the end of the shooting season, and by the following September the corneae had recovered their transparency and the vascularisation, to superficial examination, had disappeared. The method of treatment here outlined may possibly have some bearing on the idea among a certain section of the community that fasting spittle is a good eye wash.

## ABSTRACTS

### GENERAL MEDICINE

(1) **Albrich, K.—On the influence of infectious diseases in diseases of the eye. (Ueber den Einfluss von Infektionskrankheiten auf Augenleiden.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXXI, S. 61, 1928.

(1) **Albrich** makes the following postulates with regard to the influence of general infection upon diseases of the eye.

1. All diseases which tend to remove the sensitivity of the skin to tuberculin may alleviate scrofulous diseases of the eye.
2. Local infections in the vicinity of the eye (such as facial erysipelas) tend to alleviate diseases of the eye.
3. General systemic infections (such as measles, malaria, cholera) similarly affect favourably diseases of the eye, the bacterial infection tending to promote immunological activity.

Many cases are quoted in support of this thesis; such as the clearing of trachoma, scleritis, keratitis, etc., during measles, erysipelas, cholera, etc. Whether or no the treatment of intractable infections endangering vision can be justifiably undertaken on the lines of treatment of general paralysis of the insane by inoculation with malarial parasites, is a matter which should depend upon the opinion of the individual surgeon—or, perhaps, of the patient.

W. S. DUKE-ELDER.

(2) **Borgeson, E. J. and Wagener, H. P. (Rochester).—Changes in the eye in leukaemia.** *Amer. Jl. of Med. Sciences*, Vol. CLXXVII, p. 663, 1929.

(2) **Borgeson** and **Wagener**, of the Mayo Clinic, review in an instructive article the ophthalmoscopic findings in a series of 138 cases of leukaemia observed at the Mayo Clinic. Of these cases 17 were acute and 121 chronic cases, and 89 out of the 138 showed some change or other. The changes the authors observed appear to be more varied than those described in their smaller series by Leber and by Foster Moore, both of whom recognised three distinct groups or stages: (1) engorged veins, (2) engorged veins with haemorrhages and (3) engorged veins with haemorrhages and definite leukaemic infiltrations into the retina—true leukaemic retinitis. The changes observed by the authors are described as falling in five groups apart from one case in which the retinal changes were indeterminate: (1) Engorged veins, 27 cases. (2) Engorged veins with haemorrhages or exudates of no characteristic type, 35 cases. (3) Leukaemic retinitis, 10 cases: definite leukaemic or leucocytic infiltration into the retina particularly in the form of white lines sheathing the peripheral portions of the distended veins, but often also in the form of localised nodules with associated haemorrhages. The nodules have a characteristic appearance; they are round, sharply circumscribed, often elevated and are frequently surrounded by a haemorrhagic margin. Usually they are one-sixth to one-third of a disc diameter. In almost all these cases the discs were full and blurred and in some they were measurably swollen. (4) Apparently isolated retinal haemorrhages, 8 cases. (5) Retinitis of anaemia, 8 cases: " A type of retinitis which could not be distinguished ophthalmoscopically from that seen in cases of pernicious anaemia or in cases of severe anaemia secondary to

carcinoma of the stomach or colon." There is definite anaemia of the disc, dilatation of the retinal vessels, scattered haemorrhages and exudates of the superficial cotton-wool type. The diagnosis of leukaemia was established by the blood counts and other clinical features and not suggested particularly by the retinal picture.

The authors do not agree with the opinions expressed by earlier observers that the retinal changes have little prognostic significance. It is true that nothing clear emerges if all retinal changes are considered as one entity: but definite prognostic indications are available if the different changes observed are considered separately and correlated with the various subdivisions into which leukaemia is divided, *i.e.*, myelogenous and lymphatic, each of which has acute and chronic types. Furthermore it is seen that some lesions are particularly prone to occur in some special variety of leukaemia. Thus (1) retinitis of anaemia occurred most frequently in acute myelogenous leukaemia. (2) Leukaemic retinitis was found only in cases of chronic myelogenous leukaemia (in 10 out of 72 cases of this type), and in all these there was a high leucocyte count.

Reviewing the lesions in the light of the different types of anaemia the authors hold that:

(1) In the acute types of both the myelogenous and lymphatic leukaemias, the essential cause of the appearance of retinal lesions seems to be anaemia, for in these cases, as in pernicious anaemia, it is rare to find retinitis when the haemoglobin is above 40 per cent.

(2) In chronic myelogenous leukaemia retinal lesions are decidedly more likely in those cases in which the haemoglobin is low and the myelocytes are high. It is not so much a question of a high total leucocyte count as a high differential myelocyte and myeloblast count.

(3) In chronic lymphatic leukaemia the factors influencing the onset of retinal lesions seem rather less definite than in other types. The most significant would seem to be the associated anaemia.

The authors point out that the retinal exudates seen in myelogenous leukaemia are infiltrations of mature or immature myelocytes, and the myelocytes have a greater tendency to these infiltrations than the leucocytes. This explains the greater frequency of retinal exudates in cases of myelogenous leukaemia with a high myelocyte count.

On the question of haemorrhages it would appear that retinal haemorrhages may be isolated as far as haemorrhages in other tissues are concerned, in the case of myelogenous leukaemia; but in lymphatic leukaemia retinal haemorrhages indicate the presence of haemorrhages elsewhere.

As to prognosis the authors hold that in acute cases the presence

or absence of retinal changes seems to be of no significance; in chronic cases on the other hand it has some value. In chronic lymphatic leukaemia the average length of life after observation was 248 days for patients without retinitis and 72 for those with retinitis; in chronic myelogenous it was 1,039 and 314 days respectively.

By correlating the blood picture and the expectation of life with retinal changes it would appear that retinal changes occur in the stages of heightened pathological activity in the case of chronic myelogenous leukaemia: in the case of chronic lymphatic leukaemia the connection is not quite so clear.

A. SOURASKY.

(3) **Yater, W. M. (Washington) and Wagener, H. P. (Rochester). Ophthalmoscopic signs in disease of the heart.** *Amer. Jl. of Med. Sciences,* Vol. CLXXXIII, p. 105, 1929.

(3) In a study of 137 cases verified by necropsy, **Yater** and **Wagener** have investigated the significance of fundus appearances in cases of heart disease. In these investigations (conducted at the Mayo Clinic) a sharp distinction is made between the cardiac failure due to hypertension and that due to sclerosis of the coronary arteries; likewise a clear distinction is laid down for sclerosis of the retinal vessels as between the senile and the hypertension type. By the senile type of retinal arterio-sclerosis the authors characterise the changes seen as reduction of the calibre of the retinal arteries, with little or no irregularity in the lumen; arterio-venous compression is absent and the light reflex is dulled. The opposite features are shown by sclerosis of the hypertension type, the light reflex is exaggerated, while arterio-venous compression and irregularities in the lumen of the arteries are present. Retinitis is only present in cases of sclerosis of the hypertension type, and according to the severity is benign or malignant.

The 137 cases represented the main groups of heart disease, and the only changes in the fundus that are of diagnostic and practically pathognomonic significance are the embolic lesions of the retina in cases of subacute bacterial endocarditis. But apart from this, retinal changes have considerable significance in the prognosis of heart disease. This is brought out by a study of 72 of the cases in which retinal arterio-sclerosis was present.

All but three of these 72 cases of heart disease with retinal sclerosis showed hypertension; in the three in which hypertension was not present, there was reason to believe that it had existed at some time. There was an important difference in the lesions associated with the two types of retinal sclerosis. Those cases in which the retinal sclerosis was of the senile type had hypertension of only a mild degree, whereas the hypertension in the cases

of the hypertension type of retinal sclerosis was more severe and the hearts were much more hypertrophied. Furthermore all those with retinitis had severe cardiac disease, apparently due mainly to the hypertension, and all of those with the malignant type of retinitis had evidence of renal injury in addition to cardiac injury.

On the basis of their study the authors point out that:

(1) If retinal sclerosis of the hypertension type is present in a patient with heart disease, less than sixty years of age, who does not have other evident cause for such disease, hypertension may be assumed to be the cause.

(2) If in addition to the retinal arteriosclerosis retinitis of the hypertension type is present, hypertension is always the most important factor in the cardiac disease; and the course of the disease is likely to be more rapid and severe than in uncomplicated cases of retinal sclerosis.

(3) If, however, retinal arteriosclerosis of the hypertension type without retinitis is present in patients old enough to be the subject of severe coronary sclerosis, the existence of this type of fundus picture indicates only that hypertension is or has been present. One must rely in such cases on the symptoms for the interpretation of the degree of sclerosis of the coronary arteries.

(4) The hypertension associated with retinal sclerosis of the senile type is usually not severe, and if cardiac disease exists, the hypertension may be excluded as the important causal factor and severe coronary sclerosis may be assumed to be present.

(5) Ophthalmoscopic examination yields negative results in cases of heart disease unless the heart disease is due to or associated with hypertension or coronary sclerosis, or subacute bacterial endocarditis.

The article is by no means easy reading, but will repay careful study.

A. SOURASKY.

(4) **Zimmerman, L. M. (Chicago).—Exophthalmos following operation for the relief of hyperthyroidism.** *Amer. Jl. of Med. Sciences,* Vol. CLXXVIII, p. 92, 1929.

(4) Exophthalmos, though its origin is unknown, is generally regarded as one of the cardinal symptoms of hyperthyroidism, and operative relief of the hyperthyroid state is expected to cure or at any rate improve the exophthalmos. That this happy result does not always follow is well known, and now **Zimmerman** calls attention to the rather paradoxical condition of exophthalmos supervening or becoming decidedly more marked in cases where operation successfully relieved the other symptoms of hyperthyroidism.

Eight such cases are reported in detail and three more mentioned

in a postscript. In three of the eight cases the exophthalmos was unilateral and in some of the cases conjunctivitis, chemosis and oedema were present. This post-operative exophthalmos developed with a falling basal metabolic rate, and was associated in most instances with subnormal metabolism and sometimes with frank myxoedema. Thyroid medication or the withholding of it seemed to have no effect on the ocular changes.

A. SOURASKY.

## BOOK NOTICES

**Libro-Atlas de Bio-microscopia de la Córnea, iluminada con la lémpara de doble hendidura.** By J. LÓPEZ-LACARRÈRE (Madrid). Pp. 304. 35 text illustrations and 222 plates. Madrid: Blass. 1929.

The literature of the slit-lamp has been enriched by an atlas of the biomicroscopy of the cornea produced by Dr. J. López-Lacarrère, of Madrid. The volume is the first of its kind to appear in Spanish literature. The first part of the book, which comprises about a third of the whole, is devoted to a very clear and complete account of the general theory and the technique of the slit-lamp, wherein the apparatus and the principles of its use are described. The instrument of Gullstrand is preferred with the modification of the "double slit" whereby two slit-images are simultaneously produced, one with a thick L-shaped beam and the other with a thin linear beam. This was introduced by Lacarrère some two years ago (*Rev. gén. d'Ophtal.*, April, 1927) who is enthusiastic over the advantages of the double optical section, which, it is claimed, simplifies the technique and enhances greatly the sense of relief. The modification can be applied to any slit-lamp, while the examiner remains free to use either this or the old method.

The second part of the book deals with the normal cornea, and the third describes the pathological conditions occurring in this tissue, each being clearly and adequately dealt with in the text. After a bibliography which extends over twenty-eight pages there follows an atlas comprising nineteen plates depicting in colour the various conditions described in the text. Each plate is accompanied by a full clinical history which greatly increases the interest and value of the illustrations; these are uniformly good, and although they do not come up to the standard of those of the original atlas of Vogt, they bring out clearly the points of importance which are emphasized in the text. The author and the Hispano-American Society of Ophthalmology, under whose aegis the atlas is produced, are to be congratulated on the production.

**Die Radiumbehandlung in der Augenheilkunde.** By L. KUMER and L. SALLMANN. Pp. 198. 65 illustrations. Julius Springer. 1929. Price, 14.40 marks.

A valuable book on the practice of radium in the treatment of ocular disease has been written by Kumer and Sallmann. We have rarely read a treatise on a special method of therapy which presents its subject with more sound reasoning and logical and unbiassed conclusions. The tendency for most writers who sponsor a subject of this kind is to emphasize its value inordinately, to gloss over its dangers and shortcomings, and to inspire the reader with an unjustifiable enthusiasm: the authority and large experience by which the opinions expressed in the present work are backed call for more than usual consideration.

Radium has from time to time been advocated in the treatment of a very large number of ophthalmic conditions. With regard to disease of the lids the authors discourage its use in eczema, psoriasis, blepharitis, trichiasis, and related conditions. In lupus it should be regarded only as a subsidiary aid to other agencies. In fibromata and papillomata it is useless, although in small warts it is of value; in molluscum contagiosum it is valueless, but may be used, as in the event of other methods failing, in xanthelasma. In pigmented naevi it is contra-indicated, but it is the method of choice in the treatment of cutaneous angiomata: with the subcutaneous varieties results are less satisfactory. Its most valuable province is carcinoma, although the involvement of bone is a contra-indication: with its use 50 per cent. of inoperable cases may be cured, 66 per cent. of border-line cases, and 80 per cent. of operable cases. Sufficient experience has not yet accumulated to give a decisive opinion with regard to sarcomata: some types, especially the spindle-celled type, appear to be made worse. Its value in chronic conjunctivitis is doubtful, in trachoma the results are various but frequently helpful, and in spring catarrh frequently good. In pemphigus and pterygium it is useless: but in epibulbar carcinoma where an attempt is to be made to preserve vision, it may well be tried if the tumour is not too widespread. It is of no great use in diseases of the lacrymal apparatus with the exception of Mikulicz' disease.

With regard to corneal diseases too few cases have been tried to warrant dogmatic conclusions: with chronic ulcers and keratitis the results are variable, but in acne and herpes they are said to be satisfactory. With regard to diseases of the inner eye, little benefit accrues in irido-cyclitis beyond an analgesic effect, although some good has been noted in tuberculous cases. Intraocular sarcomata and gliomata are better treated by other methods, although its value is great in the after treatment to guard against or to combat orbital recurrences. In cataract and glaucoma it is useless, and in

the treatment of such conditions as myopia its use can only bring discredit to a method of therapy which in its own restricted field is of immense value.

The book deals with the general physical and biological action of radium, and outlines the methods of application suitable for each condition and the appropriate doses. Not the least interesting and important section is that dealing with the evil effects its unwise or ill-advised use may bring about. Some conjunctivitis is invariable, and this may progress, with large doses, to a radium burn. The cornea is the most frequently damaged tissue: a superficial keratitis may result, sometimes complicated by pannus formation, or an interstitial keratitis may even undergo necrotic changes, and the supervention of infection has culminated in a panophthalmitis. To guard against this a careful control by slit-lamp observation throughout a course of treatment is advised. After prolonged applications the hyperaemia in the uveal tract may result in the formation of a hyphaema or the supervention of iritis, while retinal damage involving changes in the rods and cones, the inner nuclear and ganglion cells, have been obtained experimentally. The greatest interest attaches to damage to the lens and cataract may be a late sequela, while it has been reported that the extraction of a radium-cataract has been followed by haemorrhage and an intractable iritis. The moral is that radium should be used upon the eye only with care and by an expert.

**The Eye in General Medicine.** By A. MAITLAND RAMSAY, M.D., LL.D., London. Pp. 10+255. Baillière, Tindall & Cox. Price, 12s. 6d.

This book is the second edition of the author's "Diathesis and Ocular Diseases," which came out twenty years ago and which has been out of print for many years. So many changes have taken place and so many discoveries have been made since the first edition was published that the work has had to be almost entirely rewritten to bring it up-to-date; and yet the main theme remains unaltered. More than most ophthalmologists, the author has been insistent over a great many years on the necessity for the ophthalmic surgeon to remember that in his work he must take into account the fact that eye diseases, for the most part, are due in the first instance to general conditions, and that no ophthalmic surgeon can flourish or do the best for his patients if he neglects the patient's general health. This is sanity in ophthalmology, and if it does nothing else for the practitioner than remind him that there are other causes for headache besides errors of refraction, the book will have done much good.

Each chapter is complete in itself and each contains valuable

lessons for those who can read, mark, learn and understand; the book will be a valuable addition to the libraries of the general practitioner and the ophthalmic surgeon.

**London County Council. Annual Report of the Council, 1928. Vol. III, Public Health.** London: P. S. KING & SON, LTD. Price, 2s. 6d.

The report of the London County Council for the past year contains accounts of investigations undertaken by the Council's Medical Officers and among these there are some of considerable interest to ophthalmologists.

Dr. Elizabeth M. McVail has continued her investigations into the physical and mental characteristics of myopic children. It may be remembered that in last year's report she showed that children attending myope classes have a greater incidence than normal children of signs of undernutrition and of evidence of septic and enlarged tonsils. In the present report the intelligence and cephalic indices of 110 myopic children attending myope classes are reported upon. But little support is found for the impression that prevails " particularly among people themselves myopic, that the myope is highly intelligent." The children examined were on the whole found to be a normal set, with rather fewer very brilliant ones among them, and rather more decided subnormal children. The cranial measurements recorded by Dr. McVail are of interest in view of the fact that some observers still see a relationship between cephalic indices and myopia. Dr. McVail finds that the myopes appear to have a breadth of head rather above the normal, though strictly comparable data for normal children do not appear to be available. It would, however, appear that children with the highest degrees of myopia are not the most broadheaded.

Another section of the report gives an account of the work by Sourasky on visual defects in Jewish children, published in this journal (*Brit. Jl. of Ophthal.*, April, 1928). It may be remembered that Sourasky confirmed the greater incidence of myopia in Jewish children, but did not attribute this to the excessive amount of close work done by Jewish boys in Hebrew evening classes. He held that the excessive incidence of myopia in Jewish boys was due mainly to the fact that normally Jewish children have low degrees of hypermetropia, thus lacking a hypermetropic reserve. The greater incidence of myopia in boys as opposed to girls among Jewish children he showed to be parallel to the conditions obtaining among non-Jewish children.

The conclusions reached in that paper were the subject of an independent enquiry by Dr. J. Hamilton McIlroy. Her work does not confirm the view that Jewish children have normally a lower degree of hypermetropia than the non-Jewish, thus contradicting

the view that the myopia of Jewish children does not need any explanation other than the normal process of development. If Dr. McIlroy's work be accepted then the excessive incidence of myopia in Jewish children must still be held to lend support to the theory of close work as a cause of this affection—or perhaps to some other factor operating in the environment of Jewish children. It must, however, be pointed out that Dr. McIlroy's conclusions are based on retinoscopies carried out without mydriatics, the refractions being estimated by the use of the Wright Thomson skiascope in a darkened room.

Other conclusions of interest reached by Dr. McIlroy in her investigation of 1,335 unselected cases are:

"(1) The prevailing condition at the age of seven is that of low hypermetropia—this may be taken as the normal condition of the eyes at this age, both in Jewish and Gentile children.

"(2) Hypermetropia diminishes during school life from 70 to 82 per cent. at the age of seven, to 44 to 55 per cent. at the age of thirteen. But low hypermetropia still remains the dominant refractive error at the age of thirteen and must account for the largest proportion of visual defect, even at the later school ages."

The normal changes in refraction during childhood is a subject that obviously is still crowded with problems waiting for solution.

**Government Ophthalmic Hospital, Madras. Report for 1926.**

Lt.-Col. R. E. Wright, the Superintendent of the Government Ophthalmic Hospital, Madras, has brought out his report for 1926.

He notes that dilatation of the pupil by atropine is obtained less readily in patients with heavily pigmented irides than with lightly pigmented irides. He also notes the value of adrenaline in assisting the action of the atropine. This has been found also by other observers in trachomatous countries, and has been attributed to the action of adrenaline in constricting the vessels of the conjunctiva which feed the pannus vessels, and thereby preventing the mydriatic being carried away by the blood stream, without effecting its purpose. It is also probable that the great amount of pigment in the iris tissue renders the membrane less extensible.

Col. Wright believes in a preliminary iridectomy for cataract cases, and a broad one too. He assists anaesthesia in troublesome cases by blocking the facial nerve at the stylomastoid foramen. He does not, however, refer to the occasional severe complications which have occurred from this procedure. He has some doubt about the value of retro-orbital injections of novocaine. The reviewer may state that he has used this aid to anaesthesia in all intraocular and squint operations for two years without complication of any kind.

Col. Wright discusses the danger of iritis occurring after extraction of cataract when blood or lens matter are left in the anterior chamber, and describes some research work done on iris tissue removed during the iridectomy. It would be interesting if in his next report he would consider the relation of dental sepsis to iritis after extraction. He thinks it possible that filaments of capsule adherent to the extraction wound may also cause irritation. This is certainly the case, but it would not occur if a peripheral, instead of a complete iridectomy, were done.

In a paragraph on the treatment of trachoma Col. Wright states on the authority of Kugelberg that a useful and safe remedy to place in the hands of out-patients is an ointment containing 5 per cent. of copper sulphate. It will be of great interest if he is able to report from experience if this proves to be satisfactory. A number of interesting cases are described, some of which are well illustrated with photographs.

## CORRESPONDENCE

### TRACHOMA

*To the Editor of* THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIR,—In the March number of THE BRITISH JOURNAL OF OPHTHALMOLOGY, 1928, you published a letter from me pointing out that the histological appearance of the American Indians trachoma was not the same as that found in this country. This has been confirmed in the case of Egyptian trachoma by Dr. McCartney, Bacteriologist to the Metropolitan Asylums Board, from material obtained from Dr. Rowland Wilson, of Cairo.

Dr. McCartney has also been studying the bacterium granulosis (Noguchi) obtained from the Rockefeller Institute, and has not been able to confirm any of Noguchi's work, in spite of the fact that he used the same technique and media. Neither has the bacterium granulosis been isolated from any case of trachoma in this country, although many fresh cases have been examined. A short note of this work will be found in the Metropolitan Asylums Board Annual Report of 1928-29, page 129. Investigations in other bacteriological laboratories are being undertaken in this country, but so far no results have been obtained.

Yours truly,

M. S. MAYOU.

## OPHTHALMOLOGY IN EGYPT

*To the Editor of* THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIR,—I have read with interest the article by Dr. MacCallan in your December issue, 1927, on the history of ophthalmology in Egypt. The article was most interesting, but I very much regret that Dr. MacCallan, who has done so much for Egypt, should have attacked the prominent late Dr. Eloui Pacha.

Dr. Eloui Pacha also did great services for Egypt before Dr. MacCallan, and many Egyptian oculists owe a debt of gratitude to both Dr. MacCallan and Dr. Eloui Pacha. To suggest that Dr. Eloui Pacha stooped to commercialism by benefiting financially from the dispensing of eye drops is quite erroneous. It is by no means an uncommon practice, even in Europe, to find that chemists dispense eye drops and call them after the name of some famous doctor or oculist without their permission.

This is an explanation of what happened in Egypt in Dr. Eloui Pacha's time.

Dr. MacCallan has pained many Egyptian oculists by such an unworthy suggestion of the professional conduct of a colleague who has done so much for ophthalmology in Egypt.

Yours faithfully,

NASR FARID BEY.

CAIRO,
*June 4, 1929.*

Mr. MacCallan's remarks on p. 607, Vol. XI, are as follows:

"It is interesting to remember that he ceased to be a member of The Ophthalmological Society of Egypt in 1911, as objection was raised by members to widespread advertisements issued by a drug store in which he had a financial interest."—EDITOR.

## NOTES

**Death**

WE record with regret the death on September 7 of Sir SEYMOUR J. SHARKEY, M.D., F.R.C.P., in his 82nd year.

Sharkey was an original member of the Ophthalmological Society of the United Kingdom, one of a comparatively small number of

young physicians who joined the Society at its foundation. During the early years one of the two secretaries of the Society was a physician, and Sharkey was the third medical secretary, succeeding the late John Abercrombie. He served the Society in that office for three years, taking an active part in the meetings and contributing to the papers and discussions on the medical aspects of ophthalmology. At a later period he was appointed a trustee of the Society and continued in this office until his death.

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**The International Ophthalmological Council**

THE International Ophthalmological Council have decided to hold the next Congress in Madrid in April, 1933. Professor Lundsgaard (Denmark) is the new president of the Council and Professor van der Hoeve (Holland) and Dr. Pfluger (Switzerland) are Vice-Presidents. Mr. Leslie Paton (Great Britain) is the Treasurer and Dr. E. Marx (Holland) the Secretary. It was decided that four of the eight members of the Council should retire and that they should be chosen by ballot. The lot fell to Professor Axenfeld, Dr. Byers, Professor Ovio and Professor Meller, and their places were taken by Professor Oguchi (Japan), Professor Roselli (Italy), Professor Wagenmann (Germany) and Lieut.-Col. Wright (India). Dr. de Schweinitz resigned and his place has been taken by Dr. Walter R. Parker to represent the United States. The remaining three members of the Council served on the old Council—Prof. Coppez (Belgium), Dr. Marquez (Spain) and Dr. Morax (France).

## FUTURE ARRANGEMENTS

### 1929

*November* 8.—Section of Ophthalmology, Royal Society of Medicine.

*November* 15.—North of England Ophthalmological Society, at Bradford.

*December* 3.—Midland Ophthalmological Society, at Birmingham.

*December* 6.—North of England Ophthalmological Society, at Leeds.

*December* 13.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

**1930**

*January* 10.—Section of Ophthalmology, Royal Society of Medicine.

*January* 17.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Sheffield. Subject: "X-ray Diagnosis in Ophthalmology."

*January* 20.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Leeds. Subject: "Strabismus."

*January* 21.—North of England Ophthalmological Society, address by Professor Van der Hoeve at Manchester. Subject: "Tumours and Pseudo-Tumours of the Eye and Orbit."

*February* 4.—Midland Ophthalmological Society, at Birmingham.

*February* 7.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

*February* 14.—Section of Ophthalmology, Royal Society of Medicine.

*March* 7.—North of England Ophthalmological Society, at Liverpool.

*March* 14.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

*March* 28.—North of England Ophthalmological Society, at Sheffield.

*April* 1.—Midland Ophthalmological Society, at Wolverhampton.

*June* 3.—Midland Ophthalmological Society, at Worcester.

*June* 13.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting.)

*October* 7.—Midland Ophthalmological Society, at Birmingham.

*December* 2.—Midland Ophthalmological Society, at Birmingham.

## CONTEMPORARY OPHTHALMIC LITERATURE

### American Journal of Ophthalmology. *September, 1929.*

REDWAY. Photography of the living eye: Reproduction in natural colors.
NEWMAN. Acquired axial myopia.
BERENS, LOSEY and CONNOLLY. Retroplacement for strabismus.
PETER. Important phases of a satisfactory senile cataract extraction.
JAMES. Plasmoma of the conjunctiva.
BRUNER. Cavernous hemangioma of the orbit.
C. J. and W. J. L. MCCULLOUGH. Loss of peripheral vision after head trauma.

### Archives of Ophthalmology. *July, 1929.*

FERREE and RAND. Lighting and the hygiene of the eye.
REESE. Pigment deposit in the contraction furrows of the iris.
KATZ. A localised area of calcareous degeneration in the sclera: Report of a case.
KEY. Transplantation of the temporal half of the vertical recti tendons in a case of complete paralysis of the external rectus.
FREEMAN. What does a test of visual acuity measure?
SOMMER and YASKIN. Spontaneous nystagmus. Some practical clinical features, especially its occurrence with diplopia.
POOS (trans. by ROY). The myopic problem.
GOUGELMANN. The fitting of artificial eyes, with special reference to gold ball implantation.
LANGDON. Two unusual experiences with streptococcus hemolyticus.

*August, 1929.*

DANDY. An operative treatment for certain cases of meningocele (or encephalocele) into the orbit.
JUDD. Primary squamous cell carcinoma of the cornea: Report of a case.
GLIDDON. An optical replica of the human eye for the study of the retinal image.
DEAN. Stenosis of the lacrimal ducts.
SMITH. Sympathetic ophthalmia: Report of a case occurring forty-eight years after injury.
PARKER. Management of simple glaucoma: A clinical report.
MONCREIFF. Disturbances of visual functions in concomitant convergent squint, with special reference to retinal incongruity.
LAMBERT and WOLFF. The systemic use of hypertonic solutions in glaucoma.

### The Journal of Ophthalmology, Otology, and Laryngology.

*May, 1929.*

ATKINSON. The artificial pupil as a means of restoring vision.
ROWLAND. Haemorrhage following chalazion operation on a bleeder.

### Annales d'Oculistique. *August, 1929.*

MAGITOT. The symptomatology and the problem of pathogenesis of glaucoma.
NIDA. Concerning a case of congenital ptosis operated on by a new method.
AUBINEAU. The "corneal œdema" of hypercholesterinaemia.
CARLO. Monocular perception of relief and its consequences.

### Archives d'Ophtalmologie. *July, 1929.*

CANGE. "Grains blancs" in keratitis and leprous iritis.
TRUC and DEJEAN. Median reversed V blepharotomy (bec-de-lièvre palpébral) permitting vision and closing of a defective eye.
BEAUVIEUX. The true origin of the third nerve in the cat.
VAN LINT. Some indications for tarsorraphy: Indolent ulcer, Mooren's ulcer, traumatic sclerotic staphyloma, contused perforating wounds of the anterior segment.
COLRAT. Occupational eye diseases in the artificial silk industry.
ROY. A case of monocular blindness of electric origin.

### Archiv für Ophthalmologie. *August, 1929.*

RONNE. Normal and abnormal colour systems and their mutual relationship.
NOJI. On optically produced parallel rolling movements of the eyes.
STÜDEMANN. Communication on the experimental investigation of the retarding and degenerating effect of aniline dyes.
OHM. On our knowledge of nystagmus.
SCHULTZ. Controlled production of pigmentation in albinotic eyes and sympathetic heterochromia iridis.
GUIST. The histological picture of Safir's ring line.
JAENSCH. Anatomical findings in papilloedema in myelogenous leukaemia.

KAHLER and SALLMANN. On the connection of the intra-ocular tension with the general blood pressure; also communication on the question of the law of equal stresses of Dastre-Morat.
LÖWENSTEIN and HAUROWITZ. Experimental investigations on the content of ox lenses after alteration of the surrounding medium.
ZEIDLER. Melanosis of the conjunctiva.
BLANK. Investigations of the embryonic constituents of normal cornea.
LOBECK. On the question of the source in the vitreous of alterations of its actual reaction.

## Klinische Monatsblätter für Augenheilkunde.

*August—September, 1929.*

HANDMANN. Hereditary, presumably congenital, central glial degeneration of the optic nerve with peculiar separation of the central vessels.
BIRINGER. On changes in the field of vision and the fundus in pituitary tumours.
SCHEERER. On the reason for retro-bulbar neuritis.
VELHAGEN, Jr. On the "Schauanfälle" and sleep.
CORDS. Nystagmus after injury to the skull.
IGA. On the question of sensitization and the production of "sympathetic choroiditis" with Bacillus subtilis.
THIEL. The rapidity of sedimentation of the red blood cells in ocular diseases.
RAUBITSCHEK. The present form of the arrow shadow test.
POOS. On the disturbance of the function of opening and closing the lids in Basedow's disease.
FODOR. Gonococci and inclusion bodies.
KOYANAGI. Contribution to the knowledge of primary tuberculous corneal ulcer.
KNAPP and LÜDIN. Cure of a retinal glioma with ray treatment.
LÖFFLER and WELLISCH. On the therapeutic value of diathermy for the eye.
FAZAKAS. A new point of view in the treatment of tabetic optic atrophy by means of endo-lumbar and sub-occipital pneumocephaly.
KREIKER. Operation of blepharo-chalasis with the help of von Blaskovic's lid-fold stitch.
MONASTYRSKAJA. Dynamometric measurement of the akinesia of the musculus orbicularis.
LAUBER. A space-saving modification of Bjerrum's screen and an improvement of Elliot's scotometer.
BIRCH-HIRSCHFELD. A scoop for the introduction of the fatty implant into Tenon's capsule after removal of the eye.
SCHEERER. A new entoptoscope for the observation of the circulation of the blood in the eye.
SANDER. On quantitative measurement of pupil reactions, and the simple apparatus for its use in practice.
KAYSER. A case of acquired Axenfeld-Krukenberg corneal spindle and acquired arcus juvenilis in megalo-cornea.

## Zeitschrift für Augenheilkunde. *September, 1929.*

BEHR. On the anatomy and pathogenesis of sickle-shaped degeneration of the posterior pole of the eye.
BÖCK. On our knowledge of dictyoma retinae.
FISCHER A lentoid in a case of human microphthalmos.
HEINE. Four cases of tumours of the iris: gumma, lympho-granuloma, lupoid (or sarcoid?), minimal sarcoma.
IGERSHEIMER. Affection of the optic nerve in malignant sclerosis.
JESS. The disappearance of the phenomena of copper in the eye.
KRASSO. The influence on glaucoma of general irradiation with Bucky's boundary rays.
LAUBER. The source of light in red-free ophthalmoscopy.
LENZ. On the anatomical basis of ophthalmoplegia interna.
MELLER. On the action of the ciliary epithelium on absorbing vitreous haemorrhage.
NITSCH. Neurofibromatosis of the eye.
PETERS. Concerning the affections of the eye in acne rosacea.
SAFAR. On the intra-capsular extraction of senile cataract.
URBANEK. On relapsing hypopyon iritis and its connection with tuberculosis.

### Annali di Ottalmologia e Clinica Oculistica. *June-July, 1929.*

SPECIALE-CIRINCIONE. The development of the lacrimal passages in man.
TESSIER. Note on pseudo-membranous conjunctivitis.
MORETTI. Trauma in the genesis of sarcoma of the orbit.
MARINOSCI. The ophthalmo-reactions in affections of the conjunctiva.
LODDONI. The colour sense in ametropes.
SANNA. On the connection between the volume and the behaviour of the ciliary muscle.

### Bollettino d'Oculistica. *September, 1929.*

BARDELLI. Allergic conjunctivitis.
ALAJMO. Corneal opacities from lime.
ROSSI. Clinical and histological researches on experimental tuberculous infection and re-infection of the eye.
FEDERICI. Rare episcleral granuloma due to a new species of fungus.
MARIOTTI. The clinical aspects of ocular tuberculosis.

### Archivos de Oftalmologia de Buenos Aires. *June, 1929.*

ARGAÑARAZ. Contribution to the study of functional and paralytic vertical strabismus (with torticollis). Its surgical treatment.
ADROGUÉ and SENÁ. The phenylchinconate of hexamethylenetetramine.
SATANOWSKY and SENÁ. Contribution to the study of amyloid degeneration of the conjunctiva.
FORTIN. Ciliary mucosa and glaucoma.
MANES. Transplantation of the human cornea. Operative cases.

### *July, 1929.*

BARRIEBE and MALET. Lymphosarcoma of the conjunctiva.
ADROGUÉ and PEREIRA. Voluntary unilateral movements of the eyes.
MANES. Intra-capsular extraction. Results of 150 operations.
CRÁMER and SENÁ. Intra-ocular non-magnetic foreign body.
ADROGUÉ and SENÁ. Foreign body in the vitreous.
FORTIN. The canal of Schlemm and the pectinate ligament.

### Archivos de Oftalmología Hispano-Americanos. *September, 1929.*

BASTERRA. On intra-capsular extraction with capsule forceps.
GUIRAL and VIONDI. Surgical treatment of glaucoma according to its type.
LUQUE. The syphilitic glaucoma patient.
POYALES. Fisher's simplified phakoerisis.

### Revista Oto-Neuro-Oftalmologica. *May-June, 1929.*

PEDRAZZINI. On papilloedema.

### Revista Oto-Neuro-Oftalmologica y de Cirurgia Neurologica. *July, 1929.*

KENNEPY. Ocular changes observed in epidemic encephalitis.
HALPHEN, MONBRUN and TOURNAY. Headaches in oto-neuro-ophthalmology.

### Revista Cubana de Oftalmologia y Oto-Rino-Laringologia. *June, 1929.*

RIBÓN. Albinism. The albinotic eye.
FERRER. A canula lodged in the nasal duct for three and a half years.
GUIRAL. Nystagmus and hippus.